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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

I.—ANATOMY AND PHYSIOLOGY.

(a)—COMMENTS ON THE PHYSIOLOGY OF HEARING AND THE ANATOMY OF THE INNER EAR.*†

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The literature on the anatomy of the ear and the physiology of hearing is enormous. For the most part the articles on physiology are either speculative or argumentative. All of the numerous theories of hearing have been based upon a more or less incomplete knowledge of the morphology of the ear and of the associated parts of the central nervous system. Each theory attempts to explain, so far as possible, how the recognized sensations of sound are evoked by stimulation of the ear with sound waves. Some theorists have assumed the presence of anatomical structures that do not exist, others have assigned to existing structures physical properties for which there is no observational evidence. A review of the individual theories of hearing will not be attempted today; merely to point out the weaknesses in the better known theories would require much more time than is available. Also, it would be but a repetition of what has

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been done repeatedly by the critics of each of the theories. The *status quo* of our knowledge of the physiology of hearing may, in my opinion, fairly be summarized thus: *We do not KNOW how we hear.*

In spite of considerable knowledge of the histology of the inner ear and of the brain, we are still almost totally ignorant not only with respect to how the structures of the inner ear and of the brain function when stimulated by complex sounds, such as the voice, but also as to how even the simplest sound waves, those of pure tones, are perceived. We do know that suitable stimulation of the inner ear by sound gives rise to nerve impulses in the VIIIth cranial nerve and in the pathways that lead from it to the cortex, but even for a pure tone at threshold intensity we do not know how many or which of the thousands of fibres in the VIIIth nerve are activated. Thanks to the investigations of the past few years on the phenomenon discovered in 1930 by Wever and Bray, it is well established that stimulation by sound waves causes changes in electrical potentials within the inner ear and that these cochlear potentials, as they are now usually termed, change in accordance with all the variations in pressure of even exceedingly complex sound waves. Most, but not all, of the investigators of this phenomenon are now convinced that the changes in potential occur in the organ of Corti, and probably in the hair cells of this end-organ. But it is not known whether the nerve impulses, which are entirely different with respect to changes in potentials, are directly originated by the fluctuating cochlear potentials or whether the latter are merely secondary to a basic phenomenon of which we know nothing whatever. Indeed, the idea that they are secondary is probably correct, in view of what is known in other parts of the body about the dependence of electrical potentials upon fundamental biochemical processes.

The assumption is made by most theorists that sound waves stimulate the nerve endings of the auditory nerve by causing a movement of the organ of Corti with respect to the tectorial membrane; *i.e.*, that essentially the inner ear contains a modified and very delicate form of tactile receptor. However, there is no proof for this almost universal assumption of mass movements of the organ of Corti; the idea, in any of its several modifications, is supported only by analogies

and by evidence that at best can be regarded as merely circumstantial. The same criticism holds true for the few theories in which chemical changes within the end-organ are assumed to have an important role in stimulation of the nerve endings. It is important to recall that no one has seen the living organ of Corti move under the influence of sound waves of any intensity, to say nothing of sound waves of an intensity near or at the threshold of hearing. Deductions from the behavior of models, even the least crude ones, are at best but flimsy evidence as to the actual movements within the inner ear. Equally flimsy are any deductions that can be drawn with respect to the inner ear from chemical observations in vitro. The difficulties of direct observation of the living organ of Corti are very great, encased as it is within a dense bony capsule and surrounded by fluids which are essential to its normal stimulation. If investigators eventually do succeed in overcoming the obstacles to direct observation, it is most improbable that mass movements, even if present, will be detected except under the abnormal conditions of intense stimulation. For several decades it has been well known that an extremely minute quantity of acoustic energy suffices for the threshold perception of sound. The studies made since the invention of vacuum tube circuits, etc., have but added refinements to the determinations; they have not changed the earlier conclusions with respect to the general order of magnitude of acoustic energy at the threshold of hearing. Physicists have shown that at threshold intensity, for the frequencies to which the ear is most sensitive, the sound waves advance through the air by rarefactions and condensations in which the excursions of the individual molecules are less than the diameter of the molecules. It is almost inconceivable that movements of anything resembling this small magnitude are amplified within the ear to the extent that the resultant movements of the organ of Corti can be observed even with the most powerful microscopes available.

The recent investigation by Wilska,[1] a young Finnish physiologist, is of particular interest in this connection. By a most ingenious technique he determined the movement of the umbo of the tympanic membrane in man at the thresholds of hearing for a series of frequencies between 45 and 9,000 cycles per second. Of the frequencies tested the optimum sensitivity was for 3,600 cycles; at this frequency mechan-

ically generated excursions of the umbo of only 0.0045 *millimicrons* evoked the sensation of hearing. All of the frequencies between 860 and 6,500 cycles per second were heard when the excursions of the umbo were not more than twice this distance. Transposed into more familiar terms, the excursions of the umbo at the optimum frequency tested by Wilska are 0.00000000045 cm., or less than 1/100,000th of the wave length of green light. Hearing for the lower frequencies was not elicited until the excursions were much greater than for the frequencies already known to require the least amounts of energy. Naturally the calibration of Wilska's apparatus for the shorter excursions had to be by extrapolation of the values determined by direct observation at greater intensities, so there is some uncertainty as to the correctness of his smaller figures. When transposed into terms of energy, his observations as to amplitude of movement are in good agreement with the values many observers have obtained for acoustic energy at threshold, therefore they may be accepted as of the correct order of magnitude. The work must, of course, be confirmed before final acceptance. It is, however, sufficiently correct to illustrate nicely the extreme difficulty that will be encountered in detecting movements of the organ of Corti if anyone does succeed in bringing this structure under direct observation in the living animal.

Let us now turn to a consideration of some facts about the anatomy of the end-organ of hearing. Within the time assigned it is impossible adequately to describe the entire peripheral portion of the nervous part of the organ of hearing, even if all references to physiology were omitted. Therefore, in the presentation of this phase of the topic assigned me in this symposium I shall discuss but briefly those parts of the inner ear that are not definitely known to participate in hearing.

For the higher forms of animals, those with a true cochlea, evidence is lacking to support the frequently expressed theoretical views that one or more of the vestibular end-organs have an auditory function. Phylogenetically and ontogenetically, the cochlear duct is closely related to the sacculus, and it is quite possible that in amphibians and fishes the saccular end-organs do have an auditory function. The evidence on this point may be omitted from consideration

today, because our symposium is not concerned with hearing in forms lower than mammalian. For the same reason we may omit all consideration of the structure of the vestibular parts of the inner ear. The chief reason for the recent revival of the idea that the macula sacculi of mammals has an auditory function is the failure definitely to prove that equilibrium or the responses to vestibular tests are disturbed by experimental ablation of the sacculus or by section of the major nerve to the sacculus.

The following series of lantern slides* recall to mind the structural plan of the cochlea. The names given to those parts that can be seen at the magnifications of any of these illustrations may be found in many textbooks; it is not necessary here to repeat the names or to give descriptive accounts of the size, shape, etc., of the several parts. For the purpose of our discussion today, however, it should be recalled that for the most part the cochlea consists of structures which have one or both of two primary functions: *1.* support of the peripheral parts of the neural mechanism of hearing; and *2.* conduction of sound waves to the organ of Corti.

The descriptions of the organ of Corti in most textbooks omit some details of its structure that are well established by repeated observations and that may be of great importance from the point of view of its function as a receptor organ sensitive to stimulation by extremely minute quantities of acoustic energy. The omissions on the part of the commonly consulted textbooks, combined with the difficulties of making good histologic preparations of the organ of Corti, probably account for the fact that almost without exception the writers of theoretical papers on the physiology of hearing do not even discuss the possibilities presented by movements *within* the organ of Corti. Among other omissions, most textbooks fail to make clear the relations of the outer hair cells to each other and to adjacent structures.

Each outer hair cell, of which there are about 12,000 in the human organ of Corti, is a short and almost cylindrical structure supported only at the two ends. Each hair cell is suspended, as it were, between the margins of a hole in the

*Photomicrographs of sections of human cochleae; they are not reproduced in publication because similar illustrations are readily available in textbooks. In the verbal presentation stress was laid on the essential points of the innervation of the cochlea, so far as it is known.

reticular membrane and a saucer-like expansion of the end of the cuticular process of a Deiters' cell. (Lantern slides of the organ of Corti of man and of guinea pig, also sketches on blackboard.) Except for the areas of support at the two ends, each outer hair cell is surrounded by fluid. The spaces between hair cells are small, but yet are wide enough that the cell membranes are not in contact with each other and possibly are not even in contact with the delicate phalangeal processes of the Deiters' cells which pass up between the rows of hair cells to blend with the reticular membrane. The spaces between hair cells are continuous with the space of Nuel and with the tunnel space between the pillar cells, but the fluid within the spaces of the organ of Corti is not in direct communication with the endolymph in the cochlear duct or with the perilymph in the scala tympani. Therefore, even during relatively extensive mass movements of the basilar membrane and the whole organ of Corti (if such movements do occur) the fluid within the organ of Corti would not be squeezed out, but would remain *in situ* between the cells. Mechanically the arrangement is an excellent one for permitting deformations of the cell membranes of the outer hair cells to occur either during mass movement of the whole organ of Corti or during the passage of sound waves through the intracochlear fluids. The structural pattern of the mammalian organ of Corti makes it seem inevitable that deformations of the cell membranes of the outer hair cells do occur. As to whether or not these deformations are in any manner related to the function of the organ of Corti as a receptor of sound I am not prepared to hazard a guess. However, in view of the very high degree of curvature of the cell membranes (the outer hair cells have a diameter of only 7 or 8 microns) and the facts that have been established as to electrical potentials across other cell membranes, together with the known effects of small changes in curvature upon electrical charge and the fact that relatively large changes in potentials are known to occur in the cochlea during stimulation by sound (Wever-Bray phenomenon), it does not seem unreasonable to believe that the unique structural arrangement of the elements within the organ of Corti may be related to its function.

In this connection it is of interest to note that in animals submitted to acoustic trauma—long continued loud sounds

(Hoessli)[2] or detonations (Guild)[3]—the inner hair cells of a region frequently remain intact after the outer hair cells have disappeared. The inner hair cells are surrounded by other cells instead of by fluid-containing spaces (lantern slide); this anatomic difference may account for their greater resistance to acoustic trauma. These observations may also be interpreted as evidence that deformations of the outer hair cells occur when the ear is stimulated by sound.

I have no theory of hearing to propound or to defend, and the facts that I have presented today are not new. I do wish, however, to call the attention of investigators of the physiology of hearing to the unexplored theoretical possibilities that are opened up by the excellent accounts that Kolmer[4] and Held,[5] among others, have given of the finer structure of the organ of Corti. Also, I wish to call attention to the fact that even at the present time the biochemistry of the inner ear is so completely unknown that as yet but few authors have ventured to incorporate into their theories any speculations upon possible physicochemical phenomena. This serious gap in our knowledge of the end-organ of hearing should be filled in the near future. That this be done is especially important in view of the evidence furnished in recent years as to the role of chemical reactions in the transmission of nerve impulses in other parts of the body. In the interpretation of any additions to our knowledge of biochemical reactions within the inner ear, investigators must keep in mind all established morphological facts. Structure and function cannot profitably be separated from each other in our efforts to solve the problem of the physiology of hearing.

Summary: In view of the limited time available, no attempt is made to review the enormous literature on theories of hearing or to present a systematic description of the anatomy of the inner ear. Instead, emphasis is placed on the fact that we do not yet know how sound waves cause nerve impulses to be set up in the fibres of the cochlear nerve. Attention is directed to certain details of the morphology of the organ of Corti that are omitted from most textbooks; in particular, to the relations of the outer hair cells to other structures. No theory of hearing is propounded or defended, but the suggestion is made that writers of theoretical papers on the physiology of hearing have not taken sufficiently into

account the possibilities presented by movements *within* the organ of Corti during the passage of sound waves through the fluids of the cochlea. It is also pointed out that the biochemistry of the inner ear is almost completely unknown.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

I.—ANATOMY AND PHYSIOLOGY.

(b)—THE SENSORY ENDINGS IN THE COCHLEA.*

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The general plan of structure of the primary acoustic nuclei was described some years ago (Lorente de Nó, 1933). Therefore, in the present paper only the endings of the cochlear nerve in the organ of Corti will be considered.

As is well known, the organ of Corti is innervated by the peripheral processes of the cells in the spiral ganglion. Fig. 1 summarizes our present knowledge of the subject and also includes some hitherto undescribed details.

According to the course of their peripheral processes, the ganglion cells can be divided into two classes. The peripheral processes of the cells belonging to the first class (Fig. 1, *a*), after a short course through the ganglion, run directly into the organ of Corti, grouped in small radial bundles (*r.b.*); but the processes of the other class of cells (Fig. 1, *b*) run within the ganglion for considerable distances, forming the intraganglionic spiral bundles described by Boettcher and Kölliker. The spiral bundles (Fig. 1, *s.b.*) run apicalwards, and, as established by Retzius, Cajal and Held, give off a number of thin collaterals which join the radial bundles and enter into the organ of Corti. The length of the individual fibres in the spiral bundles is not accurately known. According to Held, each fibre runs for one-quarter of a spiral or more. A similar observation has been made by the present author.

The endings in the organ of Corti can be classified in the following manner:

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1. Radial fibres (Fig. 1, *1* to *6*), ("Ortoneuren" of v. Ebner). They are processes of ganglion cells of type *a*. Upon arrival at the organ of Corti they divide into a few short branches which establish contact with the internal hair cells (*i.h.c.*). Each fibre innervates several neighboring cells, and each hair cell receives branches from a few radial fibres.

2. External spiral fibres (Fig. 1, *7* to *11*). These fibres were first observed by Retzius; however, the first exact description of their endings was given by v. Ebner. Recently Held has published a most beautiful and exact drawing.

The external spiral fibres are of about the diameter of the radial fibres. They cross underneath the internal hair cells and through the tunnel of Corti, without giving off collateral branches, and reach the level of the external hair cells, whereupon they bend upon themselves at right angles, and grouped in small bundles run *basalwards* for considerable distances.

There are as many bundles of external spiral fibres as external hair cells, but the individual fibres do not run their entire course within the same bundle, for as shown in Fig. 1 (*e.h.c.*), very often fibres of the first bundle pass into the second, and after a further traject they pass into a third one.

The length of the spiral traject is not accurately known. I have often observed external spiral fibres running through one-third of a spiral of the cochlea.

As v. Ebner has described, the external spiral fibres give off numerous side branches of small calibre, which, according to Held's description, establish contacts with as many external hair cells. My own observations also indicate that each external spiral fibre innervates numerous hair cells irregularly distributed during its traject.

Internal spiral fibres (Fig. 1, *16*). Underneath the internal hair cells there is a bundle of spiral fibres of very small calibre first observed by Retzius and recently well described by Held and Poljak.

According to my own observations, those fibres are the endings of the collaterals from the spiral bundles of the ganglion of Corti.

The fibres of the spiral bundles, before joining the bundle, often divide into numerous branches (Fig. 1, *12*, *13*). The majority of them are of thin calibre and very soon reach one of the radial bundles (*r.b.*) to enter into the organ of Corti; but more often they give off collaterals only after joining the spiral bundle (*s.b.*). Each fibre gives off a considerable number of collaterals at rather irregular intervals, and ascends toward the organ of Corti.

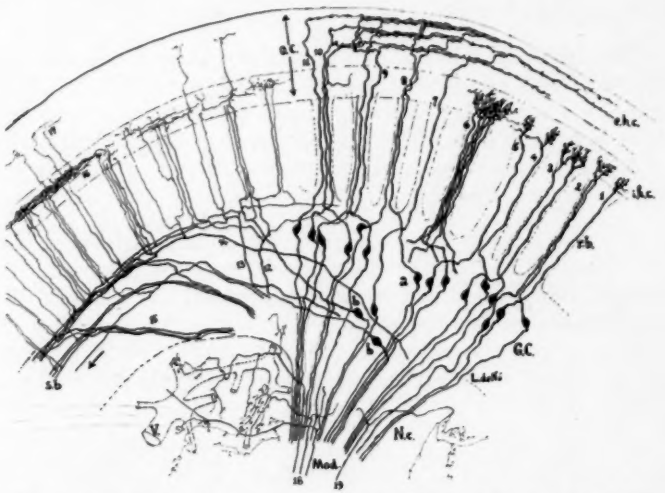

Fig. 1. Diagram of the innervation of the organ of Corti. The drawing has been made from a section through the cochlea parallel to one spiral. G.C., ganglion of Corti; Mod., modiolus; N.C., cochlear nerve; O.C., organ of Corti; V., blood vessel; **a**, **b**, cells of the ganglion of Corti; **e.h.c.** external hair cells; **i.h.c.**, internal hair cells; **r.b.**, radial bundles; **s.b.**, spiral bundles.

1 to **6**, radial fibres innervating internal hair cells; **7** to **11**, external spiral fibres innervating the external hair cells; **12** to **15**, fibres of the spiral bundles; **16**, internal spiral fibres; **17**, branches of the internal spiral fibres reaching the external hair cells. The arrow next to the spiral bundles (**s.b.**) points apicalwards.

The endings of the internal spiral fibres are not as yet well known, because the method of Golgi cannot be used in the case of adult animals. From observations made on the cochleas of six- to seven-day-old rats, it may be concluded that the internal spiral fibres innervate almost exclusively the internal hair cells, because branches reaching the level of the external hair cells (Fig. 1, *17*) are seen only very seldom.

Innervation of the blood vessels of the cochlea (Fig. 1, *18*, *19*). Recently I have been able to observe in Golgi stains of the internal ear of the six- to seven-day-old rat that the blood vessels of the cochlea are innervated by fibres arriving through the cochlear nerve.

The fibres in question leave the cochlear nerve before reaching the spiral ganglion and pass into the connective tissue which carries the blood vessels (*V*, Fig. 1). They divide into numerous branches that envelop the blood vessels with grape-like endings.

The intimate relation of the endings and the vessels has not yet been established, nor has it been possible to determine how far the nerves accompany the vessels. Such fibres have been seen only in the neighborhood of the modiolus, and it is very unlikely that they go much further; many of them positively end there.

The fibres innervating the blood vessels cannot be regarded as belonging to the cochlear nerve; they must belong to the sympathetic system and reach the internal ear through the anastomosis of the facial and cochlear nerve described in another paper (Lorente de Nó, 1925). As is well known, the facial nerve has numerous anastomoses with the sympathetic plexus of the head.

DISCUSSION.

According to the preceding description, there are three types of sensory endings in the organ of Corti, which make it possible to divide the cochlear fibres into two classes: Fibres with localized endings (radial fibres) and fibres with extensive ramifications (spiral fibres). This division is similar to that of v. Ebner into "Ortoneuren" and "Spironeuren."

The radial fibres undoubtedly conduct impulses accompanied by a very definite "local sign," but the spiral fibres cannot do so, because they may be stimulated by hair cells located at very different regions of the cochlea. Therefore, when the basilar membrane vibrates, the cochlear nerve will conduct a pattern of impulses, with components—due to the radial fibres—specific for the region where the disturbance is being produced, and other components—due to the spiral fibres—

which may be present in many other patterns. Undoubtedly the central cochlear stations will have to analyze the different patterns of impulses, but a certain analysis will be made in the cochlea itself. A possible mechanism was discussed a few years ago (Lorente de Nó, 1933).

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

I.—ANATOMY AND PHYSIOLOGY.

(c)—CENTRAL AUDITORY PATHWAYS TO THE TEMPORAL LOBES.*

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In a consideration of the auditory pathways, there should be included the peripheral receptors which have been described by Dr. Guild, the primary afferent neurons and their central terminations which Dr. Lorente de Nó has presented, and the central pathways which are extremely baffling when we endeavor to estimate their relative importance. I shall not try to present these pathways from a phylogenetic standpoint, but as they appear in the human brain. However, at times it will be necessary to outline experimental work performed on various mammals to supplement information which is available from human physiology. Kappers, Huber, and Crosby (1936) have given rather a complete treatment, from which considerable information presented here has been obtained. Ramon y Cajal (1909) described the morphology of the cells in the various nuclei, and Lorente de Nó (1933a, b) has thoroughly described the peripheral course and the central terminations of the fibres in the cochlear nerve, together with the complexity of the cochlear nuclei, with some 50 different kinds of nerve cells.

For the sake of simplicity we shall divide the cochlear nuclei into the dorsal cochlear nucleus (tuberculum acusticum and nucleus centralis) and the ventral cochlear nucleus (ganglion ventrale plus nucleus interstitialis plus nucleus posterioris plus nucleus lateralis). The dorsal cochlear nucleus is less well developed in man than in some mammals; *i.e.*, rabbit, cat and guinea pig. From the primary cochlear nuclei

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there are three bundles of fibres which leave to form secondary pathways: the trapezoid body, the intermediary bundle of Held, and the dorsal secondary fibres from the dorsal cochlear nucleus (improperly called the striae medullaris acusticae of von Monakow).

The trapezoid body contains the greatest number of secondary afferent fibres. Its fibres arise in the ventral cochlear nucleus (ganglion ventrale plus nucleus interstitialis) and pass to the ventral tegmental portion of the pons, where they course through the medial lemniscus, which is just dorsal to the basilar part of the pons (see Fig. 1).

The fibres in the intermediary bundle of Held have their origin in the dorsal portion of the ventral cochlear nucleus (nucleus posterioris), pass over the restiform body, and join the trapezoid body after crossing the median raphé dorsal to the trapezoid body. According to Ramon y Cajal (1909), the bundle is well developed in the rabbit and cat, but is deficient in man.

The dorsal secondary afferent fibres arise from the dorsal cochlear nucleus (tuberculum acusticum and nucleus centralis). They pass over the restiform body, course beneath the floor of the fourth ventricle and some of them pass to the opposite side in a decussation just ventral to the medial longitudinal fasciculus; they course ventrolaterally to the vicinity of the superior olivary nucleus and join in the formation of the lateral lemniscus. However, there are some of these fibres which do not cross, but turn back to join the lateral lemniscus of the same side (Papez, 1930). Kappers, Huber and Crosby (1936) have called attention to the fact that these fibres do not form what are called striae medullaris acustici or striae Piccolomini. The latter are made up of either arcuatofloccular paths or pontocerebellar ones.

The lateral lemniscus consists of crossed and uncrossed fibres from the dorsal cochlear nucleus, crossed fibres from the ventral cochlear nucleus and fibres of origin from the nuclei of the trapezoid body. It is doubtful if there are axons from the primary sensory neurons (Ramon y Cajal, 1909) or from the superior or accessory superior olivary nuclei. As the lateral lemniscus courses rostrally, it receives axons

from the upper and lower nuclei of the lateral lemniscus of the same and opposite sides. The neurons in these nuclei receive impulses from the neighboring tegmentum, and their axons may cross to the other side in the commissure of Probst (Kappers, Huber and Crosby, 1936). In the midbrain the lateral lemniscus courses dorsally from its position lateral to the medial lemniscus and proceeds to the inferior colliculus and the medial geniculate body. A few fibres of the lateral lemniscus pass to the superior colliculus. Fibres from cells in the central nucleus of the inferior colliculus join with those of the lateral lemniscus to pass in the inferior quadrigeminal brachium to the medial geniculate body.

The medial geniculate body in man is not generally subdivided, but Malone (1910) described a ventral and a dorsal nucleus here. Axons from these cells pass in the sublenticular portion of the internal capsule to reach the transverse gyrus of the superior temporal convolution. Poliak (1932) found that the calibre of most of the fibres in the auditory radiation was fairly large and "hardly inferior to that of the coarse somatic sensory (thalamocortical) elements. Yet there are also fibres of a medium size, and even a small number that are fairly thin." In the entire auditory radiation the absolute number of fibres was less than that of the visual radiation and of the somatic sensory one. There was no evidence for a partial crossing or recrossing of the auditory radiation in the corpus callosum to the opposite hemisphere. In monkeys in which Poliak had produced lesions involving the medial geniculate body he was able to trace the auditory radiation to the temporal cortex when it supplied only the "superior temporal convolution in its upper lip, corresponding with the lower or horizontal wall of the sylvian fossa." Only a few fibres reached the convex face of that convolution. Poliak suggests that the area receiving fibres from the auditory radiation in the monkey corresponds to Brodmann's (1909) areas 52, 41, 42 and 22, and to Economo-Koskinas' (1925) TA, TB, TC and TD. This exceeds the area of Heschl's transverse convolution. In this area the distribution of afferent fibres was continuous and free from zones lacking fibres. His experiments showed the "complete absence of other afferent fibres from the between-brain to any other portion of the convex face of the temporal lobe, a contention of some investigators."

In the human brain, Economo (1929) believes that his area TC (area supratemporalis granulosa) and TD (area supratemporalis intercalata) represents the "koniocortex of the primary acoustic sphere, into which the acoustic tract radiates from the medial corpus geniculatum." However, we have seen that Poliak's (1932) work on the monkey has indicated that the acoustic radiation supplies a greater area than TC and TD in man. The work of Poliak (1932) and Pfeiffer (1920) indicated to Lorente de Nó (1933a) "that the acoustic radiation shows a very regular arrangement of its fibres that makes an anatomical projection of the geniculate body very likely." After cortical lesions in the cat, Pennington (1937) states: "Study of the degree of secondary degeneration within the medial geniculate nucleus fails to show a point for point relationship with the auditory cortex." Ades, Mettler and Culler (1937) have shown that localized bilateral lesions of the medial geniculate bodies affect certain frequencies more than others, giving evidence that the several frequencies traverse the geniculate bodies by different pathways. The work of Lorente de Nó (1933a) and of Lewy and Kobrak (1936) shows an anatomical projection of the cochlea upon the primary acoustic nuclei, but there is no evidence of a regular projection of the primary acoustic nuclei upon the medial geniculate body.

That each cochlea has a representation in the cortex of the same and opposite sides seems to be an established fact. In cases of removal of one hemisphere in the human by neurosurgeons (Dandy, 1930, 1932; Gardner, 1933) without disturbances in hearing indicates that there must be bilateral representation. Squires (1935) has analyzed the cases bearing upon this question. He states that "a long line of investigations decidedly favors the doctrine of auditory bilateral representation." Mettler (1932) is in agreement with this point of view. Although Winkler (1911) performed experiments in cats which seemed to show that the secondary afferent fibres from the dorsal cochlear nucleus were the ones mediating impulses to the cortex, Kreidl (1914) was able to show that he could completely section the secondary crossed pathways at the pontine level in both dog and monkey, and still have hearing present. In the dog, Mettler and his coworkers (1934) have shown in a quantitative manner that the "uncrossed fibres of the auditory system are approximate-

ly equal in acoustic value to the crossed components." Bragden, Girden, Mettler and Culler (1936) have presented similar findings for the cat.

The fact that Dandy (1932) was able to remove the whole left temporal lobe in a right-handed individual without any detectable effect upon hearing indicates that this lobe is not dominant as far as the sensation of hearing is concerned. Franz (1933) pointed out the inadequacy of the concept of unilateral cerebral dominance as applied to visual apprehension and learning. The human "experiments" are far more valuable than animal ones, because in animals it is necessary to depend upon reflex responses for indicators. Although Pavlov (1928) reported that all conditioned reflexes disappeared upon removal of all the cortex, there is a great deal of evidence to show that this does not hold. For this reason it is essential to evaluate critically the indicator for the presence or absence of hearing.

While it is impossible to give a relative importance to the dorsal secondary afferent fibres and to those in the trapezoid body, it would seem that the latter would be of greater importance, particularly in man. Inasmuch as no intermediary bundle of Held is present in man, the other two crossed systems are the ones to be considered at the pontine level. On the basis of the terminal arborizations in the tuberculum acusticum, as contrasted with the terminations of Held in the ventral ganglion, Ramon y Cajal (1909) was inclined to assign conduction for auditory reflexes to the dorsal secondary paths and conduction giving rise to acoustic sensation to the trapezoid body. However, Lorente de Nó (1933b) has called attention to reasons for not assigning these roles to the different pathways. He states (p. 24): "The anatomy teaches us that there are perhaps fibres that go from the primary nuclei to the internal geniculate body without first giving collaterals to the medulla and midbrain, but also that fibres coming from every one of the primary nuclei give collaterals in the system: oliva superior complex plus nuclei of the lateral lemniscus plus posterior colliculus; since this system has connections with motor nuclei, its reflex function cannot be denied. On the other hand, it has been found that the acoustic reflexes of the muscles of the middle ear have many properties in common with the acoustic sensations (Lorente de Nó and Harris, 1933)."

The crossed and uncrossed components in the lateral lemnisci probably account for a portion of the bilateral representation. It is important to consider other crossed pathways. The dorsal and ventral decussations in the pontine region have been mentioned. The nuclei of the lateral lemniscus may give rise to fibres which cross over in the commissure of Probst. Ohnishi (1932a) has described commissural fibres which arise from the dorsal-medial portion of the inferior colliculus and pass over the midline to a corresponding place on the opposite side. The ventral supraoptic decussation (of Gudden) contains fibres which have arisen in the medial geniculate bodies to pass to the opposite sides. Furthermore, there are fibres in the corpus callosum which connect the auditory cortex of one side with that of the other. While a majority of the fibres connecting the temporal lobes are found in the portion of the corpus callosum which has a ventral position near the anterior commissure, the fibres associating the Heschl's convolutions of the two sides are found in the posterior portion of the body of the corpus callosum (Kappers, Huber and Crosby, p. 1633, 1936).

Pavlov (p. 150, 1928) reports a loss of the differentiation of the direction of sound following section of the corpus callosum, while "other differentiations were obtained in this dog easily and rapidly, but never one involving localization of the source of a sound." However, these dogs did not show other disturbances of auditory sensation. In one case Penfield and Evans (1934) found that complete removal of the temporal lobe in the human caused no disturbance of hearing, but the localizations of sounds from the contralateral side were found to be less accurate. From this discussion it is evident that there is a possibility of impulses crossing over at the pontine, midbrain, diencephalic and cortical levels. However, we cannot say what part, if any, some of these commissural pathways play in auditory sensation.

In considering central auditory pathways we usually think of the corticipetal pathways, but little attention is given to the corticifugal, probably because we know so little about their course and what they do. Ohnishi (1931) found fibres which passed from the superior and middle temporal lobes in the caudal and ventral part of the internal capsule to

reach the medial geniculate body on the same side. Furthermore, Ohnishi (1932a) described fibres which arose in the medial geniculate body and passed to the inferior colliculus

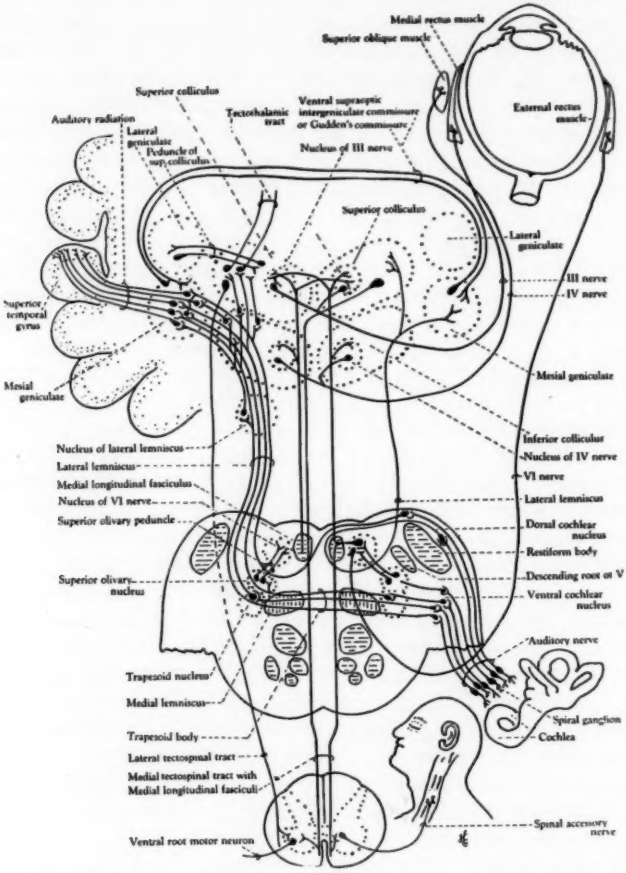

Fig. 1. Diagram showing auditory connections, including auditory conscious and certain auditory reflex paths. (Huber and Crosby, from Kappers, Huber, and Crosby, 1936.)

and lateral lemniscus, and ones which arose in the inferior colliculus and coursed downward in the lateral lemniscus. Although not all of the descending pathways can be traced,

it is rather significant that Lorente de Nó (1933b) has described centrifugal fibres which terminated in synapses on the dendrites and cell bodies of the primary acoustic nuclei. He concluded (p. 35) "that the primary nuclei cannot be considered as 'dead-beat' synapses, but rather as 'selective' synapses, which transmit impulses according to: *1.* the afferent impulses; *2.* the impulses set up in the nuclei themselves by the cells with short axis-cylinder; *3.* the impulses arriving from the upper nuclei via the centrifugal fibres." We need to know more about these centrifugal fibres with regard to their origins and courses from degeneration procedures.

In addition to the auditory pathways already considered, some brief mention should be made of reflex mechanisms, among which are some in the pons, midbrain, diencephalon and the cerebral cortex. In the pons the superior olivary nucleus receives collateral and probably some terminal fibres from axons arising in the primary acoustic nuclei of the same and opposite sides. Although it is controverted, it is claimed by some (Yoshida, 1925; Papez, 1930; and Ohnishi, 1932b) that fibres pass from cells in the superior olivary nuclei into the complex forming the lateral lemniscus. Papez (1930) believes that the superior olivary nucleus does not play the role of a reflex centre and that it gives fibres to the vestibular nerve. However, it is generally believed that the superior olivary nucleus contributes fibres to a number of motor centres; *i.e.*, the facial and abducens nuclei. From the neighborhood of the latter, impulses may be transmitted into the medial longitudinal fasciculus to reach the other motor nuclei controlling eye muscles and the neck muscles involved in turning the head.

In the midbrain the nucleus of the inferior colliculus receives impulses from fibres ascending in the lateral lemniscus. While some cells in the nucleus of the inferior colliculus send fibres into the tectobulbar and tectospinal tracts in man and some other mammals, "the major number of fibres of the tectobulbar and tectospinal system arise, at least largely, if not exclusively, in the superior colliculus" (Kappers, Huber and Crosby, p. 498, 1936). Some few fibres in the lateral lemniscus reach the superior colliculus and in addition fibres from the inferior colliculus pass to the superior one in the acoustico-optic tract.

In addition to being a way station to the cerebral cortex in the auditory pathway, the medial geniculate body is also an important centre for auditory reflexes. From it fibres pass to the zona incerta. Another pathway which is probably involved in auditory reflexes at the thalamic level is the tectothalamic tract. In four cats, which had been deprived of their neocortex, Bard and Rioch (1937) report that loud, high-pitched voices elicited a response similar to the normal expression of fear when the medial geniculate bodies were present (three cats). In one animal which lacked the medial geniculate bodies the only responses to this stimulus were erection of hair and twitching of the skin of the back. The three cats in which the medial geniculate bodies were present were able to localize a moderately soft scraping, hissing, or clicking sound. The cat without medial geniculates failed to show this reaction.

Pavlov (1928) found that auditory conditioned reflexes still continued after complete removal of the temporal lobes. When the whole cortex was extirpated all auditory conditioned reflexes disappeared. However, ten Cate (1934) found that after long periods some of the auditory conditioned reflexes were present in dog and cat after removal of the neopallium. Wolff (1937) states that the neopallium isn't necessary for the development of conditioned reflexes, but it is necessary for the development of the finer grades of discrimination.

From this brief and incomplete discussion it is evident that the central auditory pathways provide for reflex responses at different levels of integration as well as for the sensation of hearing.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

I.—ANATOMY AND PHYSIOLOGY.

(d)—VIEWPOINT OF THE PHYSICIST.*

HARVEY FLETCHER, Ph.D., New York.

It is very difficult to give the viewpoint of a physicist on this subject in 10 minutes, so I shall only give a summary of some of the facts which seem to me bear upon this question. First of all, the physicist makes measurements not directly on the neural mechanisms, but makes physical measurements of the sound waves which stimulate the ear, and then from those measurements makes deductions.

First, measurements as to the sensitivity of the hearing mechanism in terms of the sound intensities in the air were made. As Dr. Guild said, we make calculations from the measured sound intensities near the threshold which indicate that the actual displacements involved when the threshold of hearing is reached are less than a fraction of an atomic diameter. But such small displacements enable the nerve fibres or endings, or something, to start something toward the brain which is recognized as sound.

This slide (Fig. 1) shows the latest data on what you might call the limits of hearing. The lower curve gives the intensity level for the threshold at the various frequencies shown and is to be considered the lower intensity level limit. The upper limit is determined by where the amplitude becomes so large that it begins to cause damage to the tissue.

This lower curve is determined by the threshold of the nerves and the sensitivity of the transmission mechanism between the nerves and the outside air. For intensity levels about 10 dcb. below this curve there is a limit deter-

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*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 27, 1937.

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mined from physical considerations. As you know, in the air you have a lot of molecules jostling around. Now, those jostlings against the eardrum will cause the eardrum to move and, consequently, cause the inner ear to be stimulated. It turns out that those jostlings will produce a threshold inten-

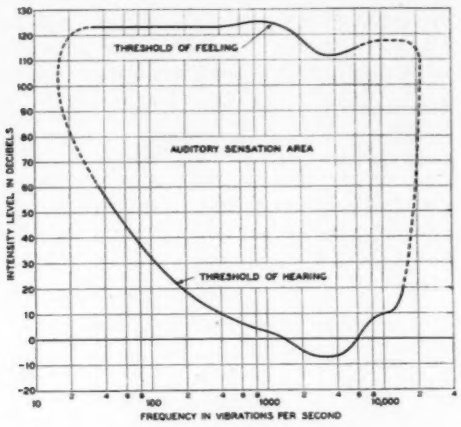

Fig. 1.

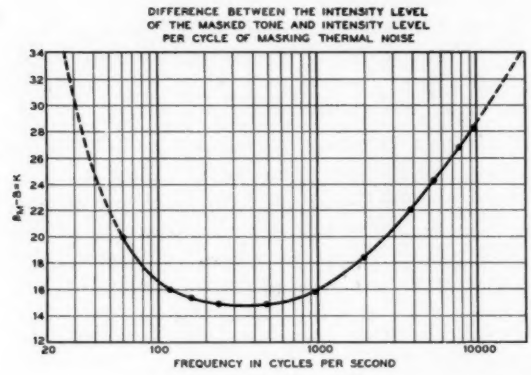

Fig. 2.

sity level just 10 dcb. lower than that for the average threshold, so very sensitive ears are actually hearing these jostlings. The hearing limits, therefore, are determined by two physical things: at the upper limit by the strength of

the tissue in the path of the sound; and at the lower limit by the necessary noise due to the vibration of the molecules.

Another measurement which the physicists can make is the masking effects of different sounds. In particular, we

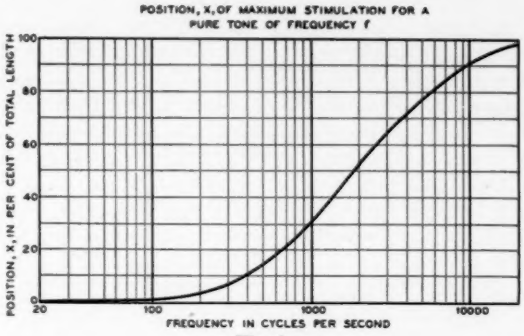

Fig. 3.

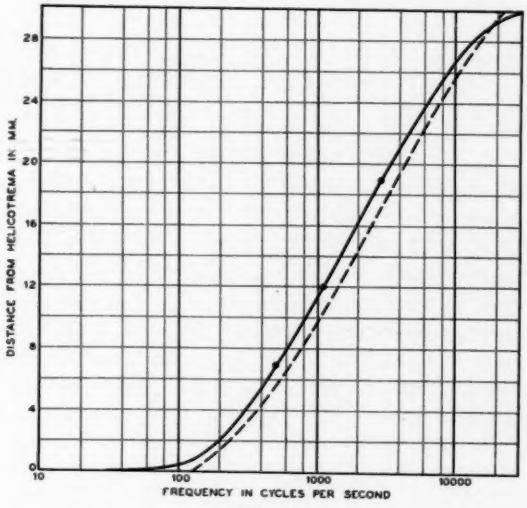

Fig. 4.

have recently been making measurements of the masking effect of thermal noise. From such measurements it is possible to determine the relationship between the position of maximum stimulation along the basilar membrane and the

frequency of the tone producing such stimulation. Let x be the position coordinate defining the position of the nerve endings. It is not necessary to know on just what kind of an established membrane these nerve endings lie, as that is the job for the anatomist. It is usual to suppose that they are along the basilar membrane, twisted into a cochlear shape having two and a half turns. In that case, the x is the distance along the membrane to the corresponding position of the nerves. In any case, the coordinate x may be considered as the per cent of the nerve endings passed over as the frequency of the tone is changed from the lowest that is audible up to the highest that is audible.

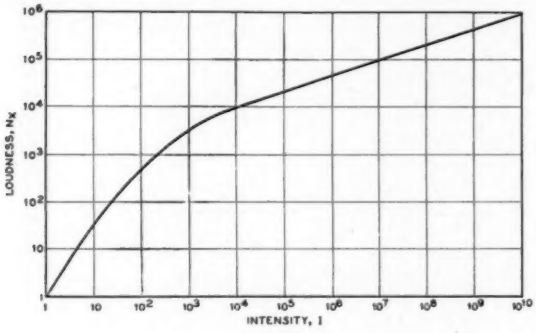

Fig. 5.

Thermal noise is a noise produced in an electrical conductor due to the jostling about of the electrons. Such a conductor may be connected to an amplifier and the thermal noise can then be raised to any intensity level. Such a noise has the important property that the power is distributed uniformly throughout the frequency range. For example, there is the same power between the frequencies 100 cycles per second and 200 cycles per second, as is between the frequencies 2,100 cycles per second and 2,200 cycles per second. Or, putting it another way, the power between 200 and 400 cycles per second is only one-tenth that between 2,000 and 4,000 cycles per second. On the other hand, the energy from the thermal noise between 200 and 400 is acting on about the same length of the basilar membrane as that between

2,000 and 4,000 cycles per second. Consequently, if masking measurements are made with undistorted thermal noise, then the amount of masking at any frequency is a measure of the concentration of frequencies at that position.

The results of such masking experiments are shown in Fig. 2. The ordinate k corresponding to the frequency f gives the difference in the intensity levels of the tone being masked and of the thermal noise. It can be shown that the position coordinate x is given by the formula—

$$x = \frac{\int_0^f 10^{-\frac{k}{10}}\,df}{\int_0^\infty 10^{-\frac{k}{10}}\,df} \times 100.$$

The assumption underlying the derivation of this formula is that a spectrum producing a constant masking would also produce a constant stimulation on the basilar membrane.

Using the data shown in Fig. 2 and the above formula, values of x corresponding to the various frequencies were calculated and are shown in Fig. 3. I wish to draw your attention particularly to the lower portion of this curve. It will be seen that from 20 cycles up to 100 cycles you are dealing with practically the same 1 per cent of the total nerves. In other words, in this low frequency region there is no position of maximum stimulation. It appears from these results that somewhere around 150 cycles per second we begin to have a definite position of maximum stimulation. In deducing the results shown in Fig. 3, it should be emphasized that they are obtained entirely from physical measurements and are independent of any anatomical measurements. If you wish to intrepret the values of x in terms of lengths along the basilar membrane, you must know how the nerves are distributed along that membrane.

Dr. Guild has made measurements on the distribution of the ganglion cells along the basilar membrane. From these measurements and the results of Fig. 3, one can deduce the actual position along the basilar membrane, in millimeters, where each frequency is sensed. Using his data and Fig. 3,

the curve of Fig. 4 was obtained. Dr. Guild's measurements make it possible to determine only the three measurements which are marked on the curve. The length of each turn was estimated by Dr. Steinberg. The rest of the curve is extrapolated from these three points. A similar curve obtained by Dr. Steinberg from a consideration of the data, both physical and anatomical, known up to the time of the publication of his paper, is given on this slide (Fig. 5) for comparison. It will be seen that the agreement is very good.

I have discussed only two phases of the contributions of the physicists to the neural mechanism of hearing, but of course there are many more which, for lack of time, I have been unable to discuss.

463 West Street.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

II.—PATHOLOGY.

(a)—THE PERILYMPHATIC AND ENDOLYMPHATIC SYSTEMS.*†

BARRY JOSEPH ANSON, Ph.D., Chicago.

The present report will deal only with those portions of the perilymphatic system and with those nonnervous parts of the endolymphatic system which are the objects of present-day research. Our new knowledge of otological anatomy and pathology is of astonishingly recent date—much of it accumulated in the short period of 10 years, and is not yet to be found in textbooks used by student and practitioner. Certainly detailed information concerning the normal structure of the internal ear is important, since in no other equally small area in the human body do slight deviations from normal produce such profoundly adverse effects upon the well-being of the individual.

PERILYMPHATIC SYSTEM.

Since the osseous labyrinth forms the confining wall for the perilymph, it would be expected to affect the contained fluid in various ways. This could be accomplished through inherent changes in the capsule, which in turn would produce alterations upon the perilymphatic orifices, either aqueduct or window; or through some less direct effect upon the vascular supply—which is primarily sent to bone.

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*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 27, 1937.

†Contribution No. 252 from the Anatomical Laboratory of Northwestern University Medical Society. A report upon certain phases of an investigation conducted under the auspices of the Central Bureau of Research of the American Otological Society.

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The perilymphatic apertures in the osseous labyrinth are the following: vestibular window, fissula ante fenestram, fossula post fenestram, cochlear window, cochlear aqueduct, and vestibular aqueduct. In the living they are occupied by a moderately vascular connective tissue; additionally, the first contains the base of the stapes, the last transmits the endolymphatic duct.

It is now a well-known fact that the vestibular window may be invaded and the contained stapes ankylosed by sclerotic bone. To the origin of this tissue in the fissular region reference will be made later; at this point we shall consider the manner of its spread and encroachment, its effect upon the perilymphatic mechanism. By the method of waxplate reconstruction (see Fig. 1) an otosclerotic nodule has been shown, both as a separate mass and, in relation to other structures, in three-dimensional form (Wilson and Anson, 1933). Histologically, the tissue presented the typical association of diagnostic features, being "richly vascularized by relatively large thin-walled vessels lodged in wide and freely communicating spaces," sharply delimited from the surrounding bone, and only mildly invasive (see Fig. 2). As a mass it was nodular and so situated that it formed the anterior margin, part of the superior and inferior margins of the vestibular window. Its spread has not yet involved the stapedial ligament or the stapes. Yet, in spite of its small size and slow rate of growth, by ultimately pressing into the vestibular window, it would have produced ankylosis of the stapes, interrupted the transmission of fluid-disturbance through the perilymph to the sensory apparatus, and have become thereby the indirect cause of deafness.

The nodule of sclerotic bone in the case just described occupied the "site of predilection"; *i.e.*, the area between the cochlea and the vestibular window; this is the area traversed by the fissula ante fenestram, which aperture we shall next consider in its relation to pathological bone.

The fissula ante fenestram, as the name indicates, is a fissure in the bony capsule situated just in front of the vestibular window (Bast, 1930; Anson and Wilson, 1933). Typically, the fissure begins internally at the point of junction of the vestibule and the scala vestibuli (see Figs. 5, 7 and 9), and ends externally at a tympanic orifice, usually placed just

below the semicanal for the tensor tympani muscle (see Fig. 6); it contains a connective tissue continuous with the periosteum of the vestibule and the submucosal layer of the tympanic cavity. But the very variety in form exhibited by

Fig. 1. Reconstruction of a sclerotic nodule which occupied anterior wall of vestibular window in an adult, tympanic aspect. Line indicates level of Fig. 2. X 14.

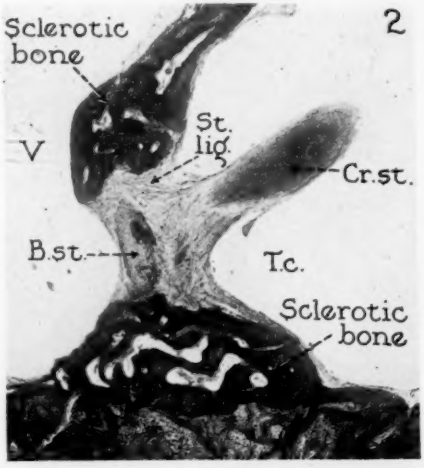

Fig. 2. Section through nodule near anterior margin of window. X 36.

the fissula in different individuals suggests that the zone which it occupies is histologically unstable (Anson and Martin, 1935; Wilson, 1935). Thus, the fissula may be incomplete, lacking one or both orifices; the extremities may vary

in relation to dependable landmarks; the tympanic orifice occasionally opening into the semicanal for the tensor tym-

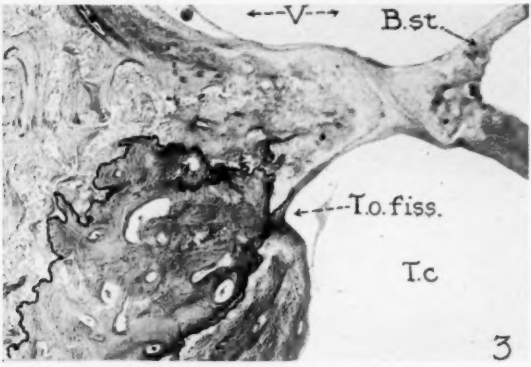

Fig. 3. Sclerotic bone obliterating tympanic portion of fissula. Adult. 18 years. X 60.

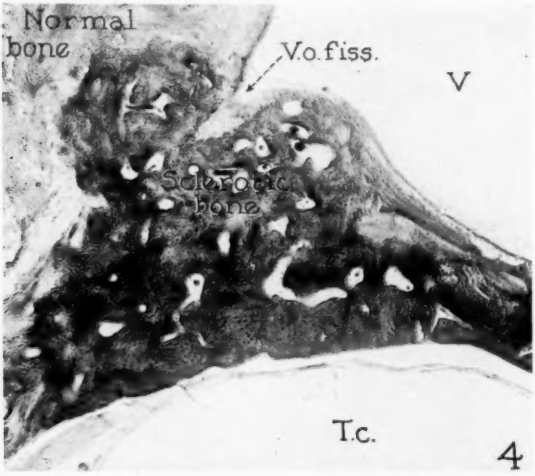

Fig. 4. Nodule of sclerotic bone obliterating all except vestibular extremity of fissula. Adult age unknown.

pani muscle, the vestibular orifice into the scala proper; an accessory orifice into the vestibular window may occur (see Fig. 8). Moreover, normal bone is not the only tissue that

remains productive in the fissular area, since with surprising frequency in young subjects immature cartilage is found in what undoubtedly was fissular space (see Fig. 10). The cartilage is still in an active state in some specimens, sending perichondrial extensions toward the fissular orifices. Occasionally, sclerotic bone in rapid development obliterates part or all of the fissular space (see Figs. 3 and 4); whether or not such bone must genetically succeed the young cartilage is a question to which we are seeking a definite answer in examining a large number of serially sectioned temporal bones. It may be said, however, that the occurrence of cartilage

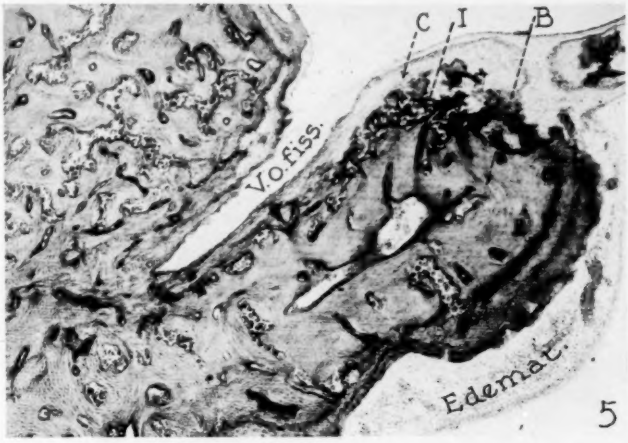

Fig. 5. Vestibular orifice of fissula. Normal form and contents. Child, 2½ years. X 54.

masses in association with sclerotic bone (within the fissular territory) supports the opinion that a definite histologic succession occurs in this histologically mobile area (Anson and Martin, 1935).

Normally, at any point throughout the length of the cartilaginous lining of the fissula, cartilage may be observed to be invaded by osteogenetic buds, the tissue converted into intrachondrial bone (see Bast, 1930); by such a process, assumedly, the fissular shell of cartilage is reduced at its tympanic extremity, the extremity itself occasionally obliterated. It may be that the newly-formed chondral masses—activated

by mechanisms now unrecognized—are later ossified in the same manner, but more dramatically since the masses are relatively large. In the bodily economy this delayed ossification may merely represent an attempt to close a fissure (Bast, 1936).

The stapes, occupying most of the space of the vestibular window, has been little studied in the past, and hence the information regarding its structure is meager indeed.* Time will not allow presentation of our recent observations.

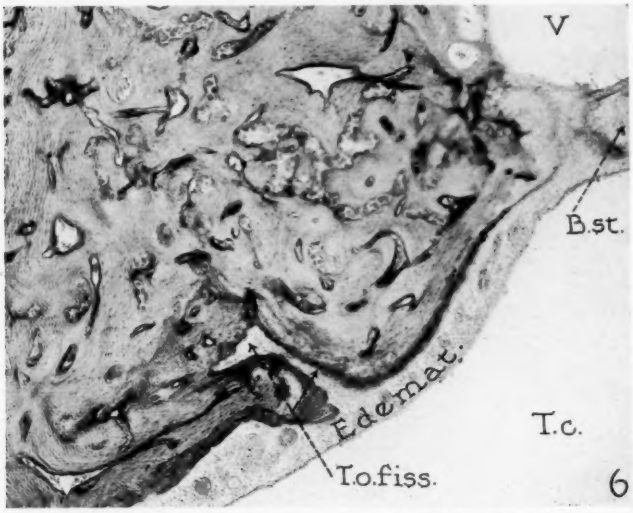

Fig. 6. Tympanic orifice of fissula. Mucous membrane edematous. Specimen as in preceding figure. X 54.

*A study, as yet unpublished, on the development of the stapes, including the histological changes which occur with advancing age. Our study is based upon waxplate reconstructions of nine fetal stages (seven weeks to term), one each of infant and child, three of adult to 70 years of age. (Abstract Amer. Assoc. Anat., Anat. Record, vol. 61, 1935, pp. 2-3). In progress also is a study of tne form of 200 excised adult stapes. At present virtually nothing is known concerning the blood-supply of the stapes—a gap in our knowledge which a current study may fill. A moderately vascular plexus is found in the submucosa, which invests its entire tympanic portion. Haversian vessels also are, not uncommonly, prominent in the bone which constitutes the tympanic aspect of the base; to what extent these communicate with the vessels on the vestibular wall (either by traversing the stapedial ligament or by passing through the islands of bone which, in older adults, reach the vestibular aspect of the base) is not known (loc. cit.); certainly the communications through the stapedial structure would in general be of slight importance since the areas of bone through which they must pass are few in number even in the 18-year-old adult and make up not more than one-half of the vestibular surface in the adult of 70 years of age. How they may be related in turn to the vessels of the adjacent fissula and fossula is as yet undetermined.

In the same category with the fissula is the fossula post fenestram, which, because it is an inconstant structure, is little understood;* so far as known, its territory does not regularly contain the variety of cartilaginous and osseous tissue present in the anterior wall of the window.

The cochlear aqueduct (perilymphatic duct) likewise belongs in the perilymphatic group (see Fig. 11). Its inner orifice is situated close to the beginning of the scala tympani at the cochlear window, its outer orifice in a depression on the lower surface of the pyramid near its posterior edge. It is said to transmit a small vein which establishes communi-

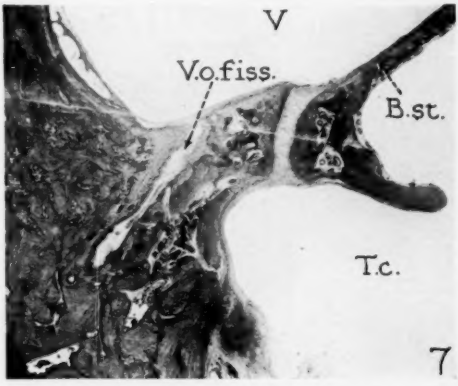

Fig. 7. Vestibular orifice of fissula. Normal. Infant, 6 months. X 30.

cation with the subarachnoid space. Actually, in some cases, it also receives a tributary from the mucous membrane, an arrangement which brings the middle and internal ear into vascular communication and provides a route for the spread of an otitis media.

Fibrotic changes in the secondary tympanic membrane (of the cochlear window) might, in reducing possible disturbance of the perilymph, reduce acuity of hearing. The contributing factors are not discussed together, or properly

*Prof. T. H. Bast informs the writer that in an examination of more than 100 series of sections, only 4 per cent of the ears possessed a complete fossula; i.e., a cleft which, without interruption, extended from the tympanic to the vestibular wall; in a much higher percentage a fossula was present in incomplete form, not infrequently occurring as a mere indentation. For the position of the fossula consult Prof. Bast's figures of the embryonic otic capsule (Bast, 1930).

appraised, and, unfortunately, time permits the merest mention of them here.

In addition to the crippling effects produced by pathological changes at the apertures, permeability of their membranes to toxic products present in the tympanic cavity may be of considerable importance; the vascular route also may aid in conveying the products of general or of distance focal infectors, or of drugs and chemical poisons, to the perilymph, therefrom to the endolymph.

Here again the course of the branches arising from larger stems in the region of the cochlear window remains to be

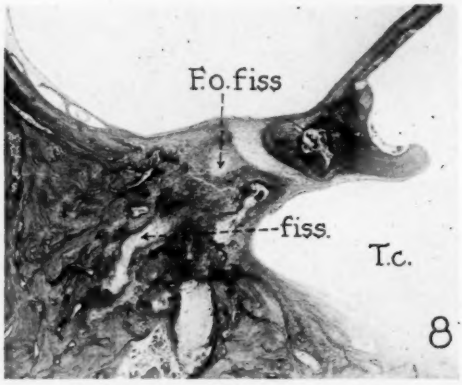

Fig. 8. Accessory (fenestral) orifice of fissula. Specimen as in preceding figure. X 30.

determined; we do not know whether the known transmission of drugs, experimentally injected into the tympanic cavity, is accomplished by dialysis or vascular transportation. In some cases at least, a vascular route is present, formed by vessels which pass into the cochlear aqueduct (as mentioned above) from each side of the secondary tympanic membrane.

The several apertures already discussed are not contiguous to the membranous sacs and tubes which contain the endolymph. Within the vestibular aqueduct, on the contrary, is contained the endolymphatic duct (of the membranous system), lodged in a relatively firm connective tissue; distally this fibrous stroma is continuous with the cranial dura mater

which invests the posterior surface of the petrous portion of the temporal bone, proximally with the periosteal tissue in the elliptical recess of the vestibule. By this tissue the medial aspect of the utricle is firmly attached to the adjacent wall of the vestibule; but on its opposite aspect the utricle is relatively free, being fastened to the other portions of the

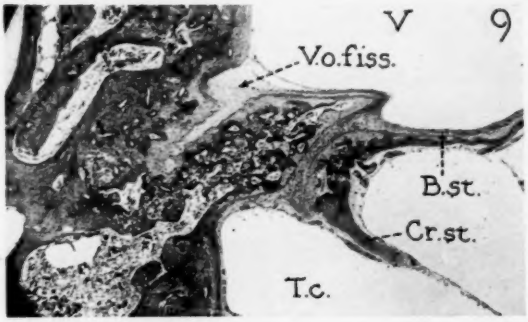

Fig. 9. Vestibular orifice of fissula. Infant, 7 days. X 25.

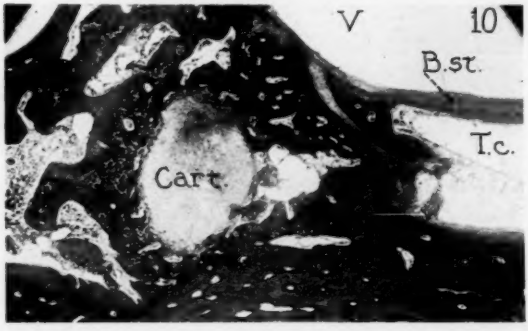

Fig. 10. Cartilaginous mass occupying fissula. Specimen as in preceding figure. X 25.

vestibular wall by very delicate trabeculae (comparable to those which tie the semicircular ducts to their enclosing canals). These bands cross the perilymphatic space and merge peripherally with the periosteal lining of the vestibule—which latter contributes the core of connective tissue for the fissula and the fossula, forms part of the annular

ligament and the secondary tympanic membrane. This continuity of fibrous cores and linings spells intercommunication of contained blood vessels.

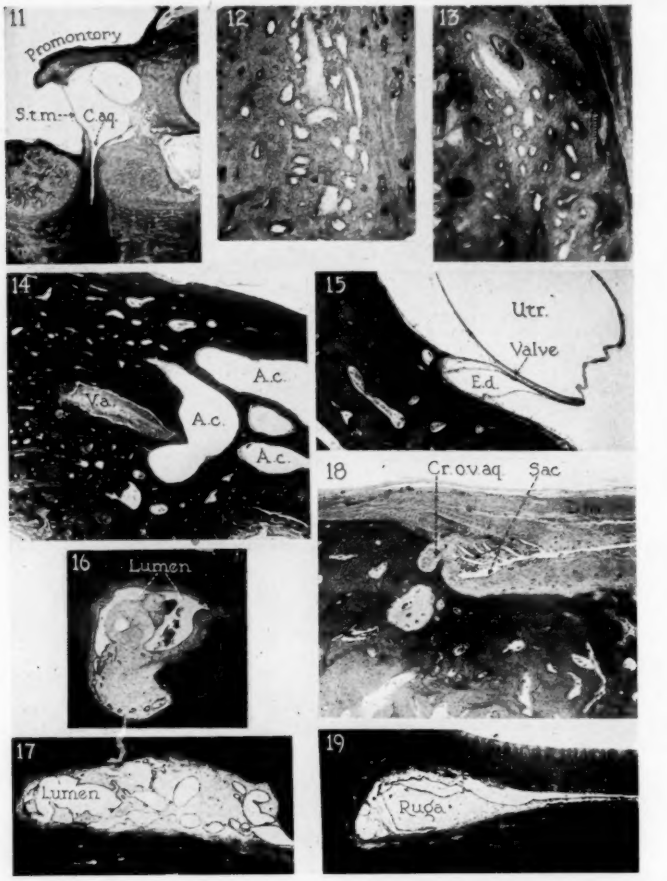

Fig. 11. Cochlear aqueduct at internal aperture. Adult, 74 years. X 48.
Fig. 12. Vascular osseous wall of vestibular aqueduct. Child, 4 years. X 34.
Fig. 13. Vascular wall of vestibular aqueduct. Adult, 50 years. X 40.
Fig. 14. Endolymphatic duct (in vestibular aqueduct) adjacent to air-cells. Adult, 68 years. X 8.
Fig. 15. Utricular valve, utricle and proximal portion of endolymphatic duct (sinus I). Infant, 3 months. X 17.
Figs. 16 and 17. Rugose intermediate dilatation (sinus II) of endolymphatic duct. Fig. 16, adult, 57 years; Fig. 17, adult, 55 years. X 19.
Figs. 18 and 19. Rugose distal dilatation (sinus III, or sac) of endolymphatic duct. Fig. 18, infant, 7 days; Fig. 19, child, 1½ years; Fig. 18, X 25; Fig. 19, X 19.

In conventional accounts describing the vascularity of the tissue surrounding the endolymphatic duct, it is merely stated that the vestibular aqueduct transmits a small vein; actually, the abundant Haversian vessels present in the wall of the aqueduct contribute to the capillaries in the connective tissue which constitutes the stroma of the epithelial endolymphatic duct (see Figs. 12 and 13); these minute vessels also communicate with a richly vascular marrow and with the vessels of the cranial dura mater; therefore, to regard the vascular relations as simple is to err seriously. Certainly the vascular route is open by which otitis media, petrositis and cerebral meningitis may involve the internal ear. And, yet, in spite of the fact that the Haversian vessels immediately surrounding, or within, the otic capsule communicate with those of the connective tissue lining the vestibule and related portions of the labyrinth, one is led to believe that the vessels of the extraperiotic area (including marrow and air-cells) must constitute an even richer set of channels. Certainly (as may be seen in naked-eye examination of sections) the bone of the periotic capsule contains relatively few Haversian spaces. It may well be that this constitutes a natural factor of safety; certainly cases are found in which a highly edematous condition of the tympanic mucous membrane is unaccompanied by changes within the labyrinth (see Figs. 5 and 6). There is supporting evidence in the fact that often meningitis, petrositis, or otitis media will produce no auditory or static disturbance.

The effects of toxins upon the fibrous tissue of the aqueduct or the vestibule are not always discoverable in histological examination, although in some specimens the hyaline nature of the stroma seems to point to pathological change. The possibility of involvement is increased in those highly pneumatized temporal bones in which the aqueduct and the vestibule lie close to air cells of the apical part* (see Fig. 14).

In the hope of throwing some light upon this important and perplexing problem we are studying the blood supply of the temporal bone in man and the laboratory animals.

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*Aware of the fact that the air cells at the so-called apex of the petrous temporal vary in size and distribution, the writer is conducting a study of those cells in individuals from different age-groups. A provisional report upon those of the child has been made (Amer. Asso. Anat., abstract. Anat. Rec., 1937, Vol. 67, pp. 4-5).

ENDOLYMPHATIC SYSTEM.

One cannot intelligently discuss the pathology of the membranous labyrinth without first giving attention to our modern knowledge of its structure. Observations show that the stylized figures of the endolymphatic and associated ducts available in textbooks are not only inadequate but misleading.

The utricular duct is not circular in cross-section, nor is its utricular orifice round (see Fig. 15). Actually, at this proximal extremity the duct is greatly compressed into elliptic form and its posterior wall is the curtain-like utriculoendolymphatic "valve" (Bast). Description of the structure was presented first by Bast (1928), as seen in the human fetus, then by Wilson and Anson (1929), in their studies of the ear of the child and of the adult.* That the valve is not limited in its occurrence to man, but is rather a regular mammalian feature, was later pointed out by Hoffman and Bast (1930), Roberts (1932), Anson (1934) and Bast (1937) in comparative anatomic observations. Evidence has accumulated in favor of the opinion that the valve does aid in closing the slit-like utricular duct (Bast, 1934; Perlman and Lindsay, 1936). The proximal portion of the endolymphatic duct, from which the utricular duct is a lateral extension, presents a sinus-like dilatation (Guild, 1927a); the lumen of the sinus in some specimens is invaded by rugae, which in miniature resemble those of the stomach (Anson and Wilson, 1936; Anson and Nesselrod, 1936). Next beyond the sinus is situated a constricted portion of the duct, which is succeeded by a second sinus-like enlargement, in which the rugosities are usually very prominent (see Figs. 16 and 17). The projections are fundamentally similar in representatives of all age groups, whether in the fetus, infant, child, young adult, or older adult. The connective tissue of the projections is more vascular than that underlying smooth areas

*The utricular valve has, beginning with the initial study carried out by Bast (1928), been described as containing blood vessels within its stroma. These are exceptionally prominent in those embryonic stages at which the valve is definitely formed (40 mm.; Anson, 1934, see Figs. 1-4). They communicate with the vessels in the mesenchymal tissue filling the vestibule. The latter vessels are for the most part lost with spread of the perilymphatic space, and, assumedly, are represented by the much less striking set of vessels within the connective tissue which lines the bony wall of the adult vestibule. A comparable change occurs in the development of the vascular supply of the tympanic cavity; the complexity is reduced as the mesenchymal tissue recedes before the advancing mucous membrane; of the abundant plexus in this region the fate of only one branch is known; namely, the stapedial artery, rendered especially striking through its unique relation to the stapes, where it is still present, at least, in the embryo of 40 mm. CR length.

of the wall, and of more delicate texture. The numerous capillaries in the connective tissue are derived, in part at least, from the vessels of the Haversian canals. In the projections capillary vessels can be traced in their coursing from the base of the elevation toward the tip, an arrangement of the blood supply which resembles that seen within an intestinal villus.

A markedly constricted portion follows the sinus, to be succeeded in turn by a third sinus-like enlargement conventionally termed the endolymphatic sac (see Figs. 18 and 19). Far from being a mushroom-shaped terminal sac, in many specimens its wall possesses a complex series of rugosities at the point of emergence from the vestibular aqueduct and a flattened prolongation within the cranial dura mater. Haversian vessels are especially abundant in the bone immediately around the aqueduct; they supply the vascular network in the stroma of the rugosities throughout the length of the endolymphatic duct; additional small meningeal vessels supply its distal portion.

Certainly the histological features are here present which one associated with the physiological mechanism of absorption; and one is therefore inclined to believe that the observations support Guild's contention (1927b), based upon experiment, that the area is one in which absorption of the endolymph takes place.*

Alteration in the histological structure would be expected to reduce effectiveness of the mechanism. In our histological studies thus far we have not found striking changes of unquestioned pathological origin; perhaps the inflammatory processes were not acute enough in the area to produce an

*As to exchange of fluid between the two systems, little or nothing is known; there is no evidence either for or against the belief that both fluids may be of common origin, passing as a transudate across the epithelial wall of the endolymphatic system; nothing is definitely known about the speed of exchange—assuming such occurs—or of the rapidity of replacement after loss.

Abbreviations used in the illustrations: a.c., air-cell; b., bone; b.st., base of stapes; c., cartilage; c.aq., cochlear aqueduct; conn.t., connective tissue; cr.or.v.aq., cranial orifice of vestibular aqueduct; cr.st., crus of stapes; d.m., dura mater; e.d., endolymphatic duct; edemat., edematous mucous membrane; fiss., fissula ante fenestram; i., introchondrial bone; sc.t., scala tympani; s.t.m., secondary tympanic membrane; st. lig., stapedial ligament; t.c., tympanic cavity; t.o.fiss., tympanic orifice of fissula; utr., utricle; v.aq., vestibular aqueduct; v.o.fiss., vestibular orifice of fissula; v.w., vestibular window.

Note regarding illustrations: Figs. 1 and 4 are from specimen described fully by Wilson and Anson (1933); Figs. 3 and 5 are from specimens shown in Anson and Martin (1935), Figs. 63 and 66, respectively; Fig. 16 from Anson and Nesselrod (1936), Fig. 22.

exudation and resultant changes, rather, affecting by diffusion the more delicate nervous elements.

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SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

II.—PATHOLOGY.

(b)—THE PATHOLOGY OF NERVE DEAFNESS.*

DR. F. H. LEWY, Philadelphia.

Six years ago, Emerson[1] wrote that the end-result in all cases of progressive deafness is nerve deafness. This, of course, is a stage of the disease at which the best diagnosis or treatment is no longer of any avail. The question, therefore, arises: What can morbid anatomy, and especially neuropathology, contribute to a better localization of nerve deafness and consequently to an earlier treatment and possibly prevention?

I will discuss very briefly the so-called congenital deafness. I omit the malformations in order to direct your attention to developmental disturbances of the spiral ganglion due to birth injuries. Albrecht[2] examined 80 stillborn children and stressed the frequency of hemorrhages into and around the VIIIth nerve. The most common locations of such intranatal hemorrhages are the porus acusticus and the point where the blood vessels enter the modiolus. In severe cases the spiral ganglion itself may be imbued with blood. Partial degeneration, insufficient development and displacement of the spiral ganglion with subsequent more or less complete deafness may be due to birth injuries. We have here a condition analogous to infantile cerebral diplegia and all we can do to prevent this cause of deafness is to emphasize its importance to the obstetricians, as we did in connection with spastic infantile paralysis.

An interesting and promising field of investigation has been opened up in the realm which we formerly designated by

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the collective term, "neuritis." It is the tendency of contemporary neuropathology to separate the exudative, infiltrative, interstitial neuritis from a purely degenerative state of the nerve, for which Wechsler coined the name "neuropathy." Under this heading the toxic and metabolic affections of the auditory nerve should be grouped. Preobrajenski[3] reported that 25 per cent to 30 per cent of the lead workers in U.S.S.R. show a considerable loss of hearing. He was also able to produce experimentally degenerative changes in the VIIIth nerve and the spiral ganglion by lead and mercury in rats. Missionjnik[4] confirmed these results in guinea pigs. It might be worth while to check these findings by the audiometer tests in workers of zinc melting plants and storage battery factories in this country. Concluding from my personal experience in the frequency of neuropathies of the peripheral nerves in lead workers, I should not be surprised to find similar changes in the cranial nerves. Such studies may offer the necessity of more effective precautions. However, this is only half of the story. There is fairly good evidence that lead neuropathy has to be considered, in fact, as a B avitaminosis—as it has been definitely proven in alcoholic neuropathy—and that it could be treated, and to a certain degree prevented, by appropriate diet or concentrated vitamin B preparations.

Halphen, Salomon and Loiseau[5] report a very instructive case of apoplectiform bilateral deafness in a chronic alcoholic, which, with an interval of a few weeks, was followed by a typical fatal alcoholic polyneuropathy. No doubt early energetic treatment in such a patient could prevent death and deafness. Diabetic neuropathy is a part of this chapter, and more therapeutic activity against the loss of hearing, so common in diabetics, might be advisable.

These considerations do not pertain to arteriosclerotic deafness. Among 26 patients which Fabinyi[6] tested with the audiometer and whose temporal bones he examined microscopically, he found an atrophy of the ganglion cells in 17, lesions in the basal coil in seven, and changes in the vessels of the internal auditory meatus in only four, whereas in Fowler's[7] experience, sclerosis is more common in the vessels of the internal auditory canal than in the cochlea.

Since we can do so little against the one form of interstitial neuritis, such as the arteriosclerosis, it is even more

important that we be prepared to apply adequately our powerful weapons against the second, syphilis. In contradistinction to the older conception, the authors agree today upon the fact that heredosyphilis plays a negligible part in the statistics of deafmutism (Locci).[8] The typical cerebrospinal syphilis and its treatment offers no new problems. This is not so with tabes. In the rather extensive literature on tabetic deafness, different theories have been aired in reference to its origin and anatomic site. In my own experience a specific tabetic atrophy of the VIIIth nerve is rare. Progressive loss of hearing during tabes seems to me usually due to gummatous infiltration of the meninges around the nerve, gradually strangulating it.[28] It is of the utmost importance to realize this situation, because it suggests the requisite of a spinal puncture in every syphilitic with progressive deafness, whether or not diagnosed as tabetic. Whenever a still active process is revealed by increase of cells or protein in the spinal fluid, immediate treatment is indicated.

The condition in the nonsyphilitic generalized infectious diseases is very similar. I touch briefly upon the encroachment of a purulent medial otitis on the acoustic nerve as a purely otological problem. In a survey of 4,650 children, Marschak[9] described unilateral deafness in 13 subjects following measles, in three each following epidemic meningitis and parotitis, in one each following typhoid and influenza. Since he found the cochlear nerve alone involved, Marschak believed in a neurogenic origin of the deafness in 26 per cent of his cases, in a meningogenic in only 6 per cent. Shambough[10] holds neuritis responsible for extreme deafness following scarlet fever and measles. However, he also states that meningitis is the most common cause of the acquired deafness—a view stressed for many years by Voss[11] and recently emphasized by McAuliffe.[12] This author mentions the purulent infiltration and degeneration embedding the VIIIth nerve in a meningitic exudate. In my own four cases of this group, the vestibular portion of the VIIIth nerve was damaged very little, if at all. The end-stage of a meningoencephalitis in typhoid is represented by a dense glial scar involving the acoustic nerve, the extra- and intrameningeal part of both the ventral cochlear nucleus and the acoustic tubercle, in which only scattered and atrophic ganglion cells

are visible. Still more dramatic are the destructions following measles. The inflammatory process almost wipes out the ventral nucleus and the tuberculum acusticum. It encroaches bilaterally upon the flocculus of the cerebellum while the vestibular nerve is comparatively well preserved. Such histological pictures, which are found in epidemic meningitis and measles, as well as in scarlet fever, typhoid and influenza,[29] should be a challenge to give more attention to the beginning decrease of hearing during these infectious diseases, and to perform a spinal tap at the slightest suggestion; first, from a diagnostic, and then from a therapeutic point of view. It seems to me possible that with better drainage the collection of infectious material in the cerebellopontile angle might be prevented.

A last group of cases of nerve deafness, in which neuropathology seems to be able to offer new perspectives, consists of the cerebellopontile angle tumors. This title covers three forms of tumors, separated by their nature and origin: *1.* Tumors of the cerebellum and the pons; *2.* cisternal arachnoiditis and tumors of the meninges, as well as of the base of the skull; *3.* tumors of the acoustic nerve. The first group consists mainly of gliomas, the second of meningiomas and of primary and secondary carcinomas and sarcomas, the third of neurinomas and von Recklinghausen's tumors, respectively. It is far from hair-splitting to differentiate between the various forms of tumors. On the contrary, they present an entirely different outlook and ask for a different surgical approach.

1a. Cerebellar tumors give cerebellar and, when they compress the medulla, cranial nerve signs. The intracranial pressure is soon elevated, but sound perception remains normal for a long time. In the case of Brunner,[13] not until 18 days before death did a relative deafness occur.—A cerebellar tumor as big as a cherry impeded the perception of low pitched tones, with full recovery after operation. Vogel,[14] therefore, assumed that the temporary diminution of hearing was due to pressure upon the acoustic nerve. Such cerebellar tumors, usually astrocytomas, give a guardedly fair prognosis to operation and are moderately radiosensitive.

1b. Pontine tumors present early paralysis of extremities and eye muscles, conjugate deviation of the eyes, sensory

disturbances and caloric inexcitability, but almost no pressure or auditory symptoms. Carruther[15] concluded from a personal observation that "little or no deafness, but ipselateral vestibular failure, strongly suggests an intramedullary lesion."

2a. The glaucomatous enlargement of the lateral cisterne, usually part of a generalized cisternal arachnoiditis (Horrax),[16] does not involve hearing. Whenever a cystic arachnoiditis is found in a patient with loss of hearing, a punctilious exploration for the tumor behind the arachnoiditis must not be neglected (Cushing,[17] Brownlie and Wishart).[18] In no one of the three groups of cerebellopontile angle tumors discussed thus far is more than a slight enlargement of the internal auditory meatus present.

2b. Meningiomas of the posterior fossa take their origin, in our present opinion, from arachnoideal cellular nests extending into the dura. Predilection places are: *a.* the transverse sinus; *b.* the porus acusticus internus; *c.* the foramen jugulare. No auditory symptoms are found in tumors of the first subgroup, while the nonauditory signs are entirely in the foreground in the last one. Only the tumors arising from the porus itself often produce early subjective and objective manifestations of the VIIIth nerve. The surgical approach of these meningiomas, through the medial or posterior fossa, depends on their fixation point. They give a good operative prognosis as long as they have not become too extensive. This, of course, is not true in the carcinomas and sarcomas of the base of the skull, which clinically behave like the meningiomas of the jugular foramen, but destroy vast areas of the osseous base of the skull.

3. The tumors of the VIIIth nerve proper have been divided by Brunner[19] in the neurinomas of: *a.* the free intracranial portion; *b.* the intraosseal part of the VIIIth nerve; and *c.* the von Recklinghausen tumors which usually are multiple.

3a. The "medial" group originating from the free intracranial portion of the nerve, unfortunately the smallest one, is the only one permitting a radical removal of the tumor. In these tumors subjective and objective cochlear symptoms are late, not only in comparison with increased intracranial pres-

sure, but also with the appearance of vestibular signs. Even with progressive loss of hearing, more or less extensive auditory rests are demonstrable. Cushing stresses the fact that the medial neurinomas of the VIIIth nerve produce first brain and later auditory symptoms. Brunner[20] saw in two large tumors of this group, compressing the medulla and the cerebellum, respectively, despite considerable atrophy of the sensory end-organs, not more than a slight loss of hearing. These patients infrequently outlive the increasing intracranial pressure to become completely deaf (Brunner). When the degree of hearing varies considerably over a short time, the deafness should not necessarily be considered to be caused by degeneration of the nerve-ganglion apparatus of the cochlea. It may be due to a concomitant hydrops, a choking of the VIIIth nerve, analogous to the choking of the optic nerve. The medial neurinomas, as the meningiomas, do not enlarge the internal auditory meatus, but produce exostoses in it and in its neighborhood.

3b. The "lateral" neurinomas of the VIIIth nerve, taking their origin from the nongliose; *i.e.,* distal portion of the nerve, are occasionally of minimal size and then represent an accidental finding at autopsy. The larger lateral neurinomas cause early cochlear and vestibular nerve dysfunction. Deafness is usually complete many years before intracranial symptoms appear. These tumors grow in the direction of the meatus, which becomes considerably enlarged. Later on the tip of the pyramid also is destroyed. Gradually the tumor invades the entire temporal bone, absolutely sparing the internal ear, however. Finally, it perforates the tegmen tympani and indentates the temporal lobe of the brain; or it grows downward and compresses the nerves in the jugular foramen or erodes the carotid canal. The VIIth nerve may be involved, though infrequently completely paralyzed by the medial as well as by the lateral neurinomas. The former attack the nerve within the cranial cavity, the latter in the Fallopian canal. In cognizance of these peculiarities of growth, the attempt to remove radically such a tumor is obviously condemned to failure. Cushing, therefore, confined his effort to decompression and to repeated reduction of the tumor by scooping it out piecemeal with the Bowie. In addition, the occasional erosion of the carotid canal should warn surgeons, not so skillful as Dandy, to remove completely an adherent

tumor capsule. It is true that the operation is palliative; on the other hand, these tumors grow extremely slowly and the surgical intervention, incomplete as it may be, assures the patient a fairly comfortable life, even over decades.

3c. The neurofibromas of the von Recklinghausen type give the least favorable prognosis, not only because of their multiplicity, but for their relative malignancy. They rise from the final distributions of the cochlear nerve as high up as the superior whorl and destroy the internal as well as the medial ear (deKleijn and Gray).[22] Bilateral deafness and deficiency of labyrinthine function are the characteristic signs of these tumors. The destruction of the temporal bone usually precedes the enlargement of the internal meatus.

Due consideration of the characteristics of the various aforementioned cerebellopontile angle tumors will contribute to an earlier and more correct diagnosis and localization, and to a more effective surgical approach.

In contradistinction to the marked progress in the conception of nerve deafness from diseases of the extramedullary part of the VIIIth nerve, our knowledge of nerve deafness from brainstem and cortical lesions has advanced little since Siebenmann[23] and Christ described their famous case of a huge pinealoma of the tectum and tegmentum 40 years ago. Most of the cases reported since lack either exact audiometer tests or are void of a serial examination of the brainstem or of the temporal bones. In other patients in whom all these conditions were fulfilled, Brunner[24] found an atrophy of the spiral ganglion and of the basal coil. He speaks of a descending degeneration transgressing the primary acoustic centres. However, this explanation requires further confirmation.

Word deafness is certainly more of an agnostic and alectic disturbance (Ranschburg,[25] Bárczy[26]) than due to a defect of perception in the tones from b' to g^2, Bezold's language text. Not one of the often quoted cases of cortical deafness has been adequately examined clinically. Bonhoeffer,[27] on the other hand, could prove that preservation of a small fraction of the deepest part of Heschl's convolution on one side guaranteed bilateral perception of the whole scale of tunes.

A much closer systematic cooperation of otologist, neurologist and pathologist is necessary if the problems of nerve deafness still open to discussion are to be ultimately clarified.

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3400 Spruce Street.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

II.—PATHOLOGY.

(c)—PATHOLOGY OF THE ORGAN OF CORTI.*

DR. M. H. LURIE, Boston.

Correlation of pathological changes in the organ of Corti with the hearing loss of animals gives definite clues as to requisites for the prediction of functional loss from examination of histological sections.

Changes present in the stria vascularis, cells of Hensen and Claudius, give no information as to the amount of functional loss.

The loss could only be determined when there were definite changes in the hair cells of the organ of Corti.

The external hair cells degenerated or showed pathological changes before the internal hair cells did. With the internal hair cells present and external hair cells degenerated, there was a partial functional loss. With both the internal and external hair cells degenerated, there was a complete functional loss.

As a result, the emphasis in our examination of the organ of Corti has been on the presence or absence of the hair cells only. If the nuclei of the internal and external hair cells can be made out, normal function is assumed for these cells, and with their absence, loss of function. On this basis definite predictions have been made as to the functional loss present which have been found to approximate closely the electrical audiogram of the cochlea.

Various animals deaf from operation on the cochlea, long exposure to intense tones, chemical poisoning, and the con-

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genitally deaf have been studied from this viewpoint. In all these types the prediction of functional loss has been found to agree with the electrical audiogram.

In the congenitally deaf waltzing guinea pig that gave no electrical response, no hair cells were present in the organ of Corti, though the other structures, such as Reissner's membrane, stria vascularis and tectorial membrane, appeared normal. Some of these animals showed an interesting transition from complete absence of the organ of Corti in the basal whorls to an almost normal organ of Corti at the helicotrema. In these animals response was obtained up to 500 c.p.s. at high intensities.

Several animals tested electrically showed losses from 30-40 dcb. throughout the hearing range. Histological examination showed degeneration of the external hair cells of the organ of Corti. The cause of this degeneration was not known and was probably secondary to some disease.

A number of guinea pigs, made partially deaf by exposure to intense tones for long periods, also showed definite degeneration of the external hair cells and occasional internal hair cells. These animals showed loss of function by the electrical audiogram from 20-60 dcb., depending on the amount of degeneration present.

Cats (including Dr. Dworkin's) operated on, to produce deafness, showed similar changes. The predictions made from the histological examinations checked very well with the electrical audiograms.

Hemorrhage into the scalae may cause deafness by physical interference, but the amount of functional loss could not be estimated from our examinations. The collapse of Reissner's membrane with normal organ of Corti present also presented difficulties in the determination of functional loss, as this also appears to be a physical interference. We have found that with this condition present the electrical test gives harmonics for the region involved, shown by marked distortion of waveforms as seen by the cathode ray oscillograph.

The pathological changes in the spiral ganglia and cochlear nerve have not been mentioned, because experiments on the correlation of the animal's hearing loss with the changes in

the spiral ganglia, cochlear nerve and organ of Corti are now being carried out.

The attempt to estimate the amount of functional loss of the organ of Corti can only be done on the pathology of the hair cells. This loss should be estimated on the presence or absence of the external hair cells, or the presence or absence of both external and internal hair cells. We have not found external hair cells present and internal hair cells absent in any of the animals examined.

With loss of the external hair cells a functional loss from 30-40 dcb. can be estimated. With absence of both external and internal hair cells the loss may be estimated from 60-90 dcb., depending upon the extent of the lesion. The larger the lesion, the greater the intensity of the sound needed to spread over the gap present. It is, therefore, important that in testing animals stimulation at the threshold should be used rather than maximum stimulation. Maximum stimulation will cause any normal or partially normal organ of Corti to respond, giving an end-result that cannot be interpreted in terms of the pathology found in the organ of Corti.

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MOTOR DISORDERS OF THE CENTRAL NERVOUS SYSTEM AND THEIR SIGNIFICANCE FOR SPEECH.

PART II.—CLINICAL FORMS OF MOTOR DEFECTS (THE "SPASTIC CHILD").*

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The brief discussion of dysarthrias of speech in the first part of this paper should serve as a suitable introduction for this presentation. It is my purpose in this second part to deal with the various clinical forms of early motor disturbances of childhood known at present under the collective name, "the spastic child."

"Spastic child" is an unfortunate and confusing misnomer. It applies to, and is descriptive of, only one—true, the largest —group of motor disorders of early childhood. To increase the confusion the term hardly does justice even to this group. It selects one characteristic only and ignores the many existing clinical variations. The result of the injudicious use of the term "spastic child" is, that as a rule, all forms of motor disorders are thrown together, that actually have nothing common with "spasticity" in the neurological sense.

It seems highly desirable that the existing chaotic terminology be clarified. Without any doubt all specialties interested in the motor disorders of childhood, and above all, the speech teacher, would be greatly benefited by such a development. It is hardly conductive to constructive work when, according to everyday experience, vastly different problems, like athetosis, cerebellar lesions, and even mental defect, are in a haphazard fashion classified together under the heading of "spastic child."

Progress in this field will begin when the terminology used by the various allied specialties will be brought into complete harmony with correct and up to date neurological concepts. It is suggested here, that the use of the all inclusive

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term, "spastic child," hereafter be dropped completely. Its place should be taken by the term, "motor defective child," or "motor deficiency." This seems to be highly acceptable, in analogy with terms that are in use at present, like "mentally defective child," or "mental deficiency." Under such a correct main heading the various forms of motor defects may be clearly and conveniently classified.

CENTRAL MOTOR DEFECTS—(BIRTH PALSIES).

1. Pyramidal lesions (Spastic paralyses): Monoplegia (paresis), Hemiplegia (paresis), Paraplegia (paresis), Diplegia (paresis), (Tetraplegia).

2. Extrapyramidal lesions (Hypermotility): Hamiathetosis, Bilateral athetosis (Athetose double).

3. Cerebellar lesions (Ataxia disorders): Hemiplegia, Diplegia (Tetraplegia).

As the above table of classification suggests, the discussion in this paper will be limited to motor defects that are due to antenatal or natal factors and consequently are present at the time of birth. These early disorders have particular significance for the speech teacher, inasmuch as they influence decisively the acquisition of motor functions and the development of speech.

A few brief words as to their causation may be desirable here. Antenatal factors are: *a.* developmental, *b.* fetal disease of the C.N.S. The development of the central nervous system may be interfered with at any stage due to some fault in the anlage or due to some mechanical cause. The result will be a malformation of some part or of the whole of the brain. If the defect is in any portion of the three motor systems, we are dealing with some form of motor disorder. Intrauterine infections may produce a fetal encephalitis and may lead to a more or less extensive destruction of the C.N.S. The resultant clinical picture will depend on the site of the lesion and on the interference with further growth of the brain.

Natal factors are largely represented by accidents caused by the mechanism of birth. The molding of the head during birth may cause an injury to a blood vessel in the intra-

cranial cavity. Such an injury results in a hemorrhage which leads, on the other hand, to a destruction of tissues of the C.N.S. Dependent on the site and the size of the destruction, we may have before us a defect in any one of the three motor systems.

Whatever the nature of the pathological process might be, the resultant clinical and neurological picture may be exactly the same. However, antenatal factors, as a rule, will lead to more decisive defects the earlier in fetal development they become operative.

Functionally any defect will alter in a wide sense the entire C.N.S.; it affects, as a matter of fact, the whole organism. However, the nature of the problem under discussion makes it imperative, particularly from the viewpoint of the speech teacher, to determine as closely as possible, how far the defect is limited to the three motor systems. It is essential to answer the question whether the general intelligence of the child is in any way damaged or not. This answer often presents an extremely difficult problem. The motor defective child is so vastly different in most instances from a normal child, that it may easily create the impression of mental defect, although its psychic functions are largely intact. The present methods of psychological examinations are still very unsatisfactory. Still the speech teacher hardly may start to work with any assurance on a motor defective child without having this important information on hand.

The motor defects found in a given child are not always clearcut. They may be a combination of disturbances in more than one motor system. Here again the speech teacher will be greatly aided by correct localization, diagnosis, and prognosis.

PYRAMIDAL LESIONS.

They are also known under the name of spastic paralysis, birth palsy, Little's disease, etc. The extent of involvement of the muscles of the body forms the basis for classification in the table above. If only one extremity is involved, it is monoplegia; if one-half of the body is affected, it is hemiplegia; if the two lower extremities are paralyzed, the name is paraplegia; and finally, if all four extremities are stricken,

it is called diplegia (or tetraplegia). "Paresis" is used to denote the less complete involvement of any part.

The lesion, whatever its nature may be, is localized either in the cortex, or in the subcortical areas of the parietal lobe of the heterolateral hemisphere. A lesion in the dominant hemisphere, where the highest centers of speech are represented unilaterally, will have much greater significance for speech than a similar lesion on the other side. Inasmuch as the cortical areas of the lower extremities are at the topmost part of the parietal cortex adjacent to the sagittal fossa, it is evident that paraplegia will be accompanied by speech involvement less frequently than monoplegias of the dominant upper extremity or hemiplegias of the dominant side. The more distalward the lesion extends in the parietal lobe, the more likely it will involve the cortical areas of the cranial motor nerves, and thus affect speech. The more it extends toward the frontal lobe, the more are the chances for including the so-called motor "speech center" and complicate matters in the sense of aphasia.

An important contribution to the understanding of athetoid movements complicating spastic paralysis comes from the work of the Yale School of Physiology. They have shown experimentally that athetosis may be produced by lesions in the frontal lobe. It seems that when the lesions of the parietal lobe extend into the upper convolutions of the frontal lobe, we may have before us spastic paralysis with athetosis.

Often in hemiplegias we see an almost complete absence of "spasticity." This form of lesion has its site frontalwards to the motor areas of the parietal lobe.

In other instances definite trophic changes in the extremities may be present. In such instances the hemiplegic side is retarded in growth and remains considerably smaller than the unaffected side. Particularly may this be true of the upper extremity. It is not quite clear where this trophic factor in such a lesion may be localized.

Spastic paralysis is characterized by the absence or decrease of conscious voluntary innervation in the affected muscles. The tonus is from slightly to enormously increased, the balance of agonist and antagonist muscles is greatly disturbed. This situation leads to a predominance of the more power-

ful muscle groups and produces functional changes, like "scissors position" of the legs, shortening of the tendo Achilles. These phenomena are always more pronounced on attempted innervation or on other stimulation. The deep reflexes are greatly increased and often there is "clonus" present. There are also certain pathological toe-signs to be found, like the Babinski or Oppenheim sign.

These latter ones are considered as characteristic for pyramidal lesions.

As already mentioned above, occasionally spasticity may be entirely absent, although the other neurological signs are present. In such cases, instead of the usual "scissors gait" and tiptoeing, there is circumduction of the leg, and dragging of the foot when the child begins to walk.

It is almost unnecessary to state that in all forms of pyramidal lesions there is a delay in the development of motor functions. Sitting up, standing up, walking, etc., may develop one or two or more years later than expected physiologically. When the upper extremities are involved, their use may remain more or less completely lost forever.

Speech development, as a rule, will be somewhat restored, even in those cases where there is no direct involvement of the pathways for this function. The delay, of course, will be more decisive in every case, where such an involvement is present.

Whenever the lesion is such that an aphasia element complicates the problem, the development of speech may be even more retarded. The question, whether in a given child we are dealing with aphasia, or aphasia combined with dysarthria, or purely with dysarthria, is not only an academic one. It actually has very great practical and clinical importance. Unfortunately, the answer may not be easy.

The type of speech we find in a child with spastic paralysis —if speech has already reached a satisfactory development— is the same as described in the first part of this paper under dysarthrias due to lesions of the corticobulbar tracts.

Motor development in these children with pyramidal lesions, inclusive of speech, takes place as a result of growth and maturation of the C.N.S. It is well to bear in mind—reempha-

sizing the fact—that in case of destruction of any ganglion cells, no regeneration is possible, and that no lost functions of the pyramidal ganglia can be taken over by other parts of the hemisphere. Therefore, in any form of motor development or compensation in these handicapped children we are dealing with residual functions. All our work with these children—remedial or training in nature—cannot accomplish more than to bring the residues to their highest potential development. For a speech teacher to possess such realistic attitude is extremely wholesome. After all, no form of training may claim to be anything resembling a creative process. We are aware of our limitations only too well in the training of normal children, and must reorganize our limitations in an increased degree with children who start out necessarily with a very serious handicap.

It is well to keep in mind, when we think of remedial teaching of speech, that any child with a spastic hemiplegia or diplegia may learn to walk in a fashion without the slightest outside help, but it never will be able to use the hand or hands for any of the physiologically intended fine functions.

In the case of language where, after all, the entire C.N.S. and even the entire person is involved, of course, the problem is more complex than in the use of the extremities. No satisfactory answer has been given as yet to the question whether, in a case where the dominant hemisphere is involved in spastic paralysis, the other hemisphere may in any way compensate for the loss of speech. The problem always remains extremely difficult in birth injuries, because we never know which hemisphere was originally the dominant one.

The motor defective child—the spastic child—will always remain defective. Even though it is able to speak fluently, if the use of the upper extremities, or that of the dominant hand is eliminated, its language in the sense of the total function of the C.N.S. and the total participation of the person will be incomplete.

EXTRAPYRAMIDAL LESIONS—(ATHETOSIS).

It was mentioned above that certain lesions of the frontal lobe may cause athetoid movements. This form of athetosis usually appears in combination with pyramidal lesions or spas-

tic paralysis. In these children we find the evidences of a more or less severe form of spastic hemiplegia or diplegia—occasionally a monoplegia of an upper extremity. In addition to these phenomena, there are evident in one or more extremities, involuntary and uncontrollable movements. As a rule, in such cases the upper extremity is more severely affected than the lower extremity. In these cases we are dealing with a combined involvement of the pyramidal and extrapyramidal motor systems, and therefore the problem becomes more difficult to handle from every practical point of view. What will be said in the further part of this discussion about athetosis, applies to this part of the problem with certain modifications to suit the specific situation.

That lesions of the frontal lobe should be responsible for such athetoid movements in intracranial birth injuries seems rather plausible, inasmuch as the cortical representation of the extrapyramidal system is in the frontal lobe.

A much more important and more serious problem that so often confronts us in the form of an extrapyramidal lesion has been known for a long time under the name of congenital bilateral athetosis (athétose double of the French literature). As the above name already suggests, here we are not dealing with the results of an intracranial birth injury, but something more fundamental. Although theoretically a bilateral birth injury of the frontal lobe might be able to produce a somewhat similar clinical picture, actually such lesions do not occur. On the other hand, in congenital bilateral athetosis no lesions of the frontal lobe have been found.

In these cases we are dealing with a developmental problem, apparently an agenesis or dysgenesis of the extrapyramidal system. The part that seems to be chiefly affected is the caudate nucleus (striate body) unilaterally or bilaterally. Although the entire pathophysiology is not yet fully clear, it seems plausible that such a lesion may account for the entire symptom complex.

In congenital bilateral athetosis usually the two sides of the body are equally affected, although at times one side may present a more severe involvement. The picture is dominated by more or less wild uncontrollable primitive motions of all four extremities. These motions are suggestive of defense

movements, or climbing motions of the large muscles, and slow worm-like motions of the fingers and toes. The face usually is distorted by violent "emotional" innervations. The child usually is very unstable emotionally and may easily go from one extreme of emotion to the other.

The muscle tonus usually is decreased, although temporary "fleeting" spasm may occasionally give the impression of "spasticity." No increase of the deep reflexes is present and the pathological toe signs are missing.

The motor functions of any kind are very greatly interfered with due to the involuntary movements, which may become more accentuated on intended innervation or on any other stimulation. The result of this is that motor development in these children is much delayed, and the more severe forms never learn to sit up, stand, or walk.

Speech development is very much retarded. It seldom reaches out of that stage which was described in the first part of this paper. This is easily understandable in view of the fact that the involuntary movements, as a rule, extend to all the muscle groups that are needed for physiological speech.

The unfortunate situation for the speech teacher exists in the fact that these children—with the exception of the very mild forms—hardly ever learn the control of involuntary movements and thereby are permanently incapacitated. The other serious difficulty arises from the emotional instability of these children, which makes their training an extremely difficult task.

Often these children give the impression of low mentality, although actually they may be of average, or even brilliant mentality.

The greatest practical significance of congenital bilateral athetosis is in the unfortunate fact that it is seldom correctly recognized and diagnosed. These athetoid children are falsely labeled as "spastic" and then the great disappointment follows because they do not make any progress. They must be classed separately because not only their prognosis is more serious, but the entire approach to their problems must be vastly

different. The methods of orthopedics, physiotherapy, etc., that are applicable to the child with spastic paralysis are useless and contraindicated in these children.

The psychological approach and the best methods of training by the speech teacher still await their development, but I rather doubt that they ever will bring any tangible results for these unfortunate children.

CEREBELLAR LESIONS—(ATAXIC DISORDERS).

These are the problems which arise from intracranial birth lesions in the posterior fossa above the tentorium. Hemorrhages in this area may destroy some part or the whole of the cerebellum. The loss of function of this important organ of equilibrium, coordination, and tonus regulation leads to poor use of the muscles, dysmetria, awkward ataxic movement, dysdiadochokinesis, disturbed balance. The gait is on a broad base and rather unsteady. The muscle tonus is decreased, but there is no difficulty in voluntary innervations. The reflexes are unchanged.

The various disturbances of speech arising from cerebellar lesions are enumerated in the first part of this paper. Children with such lesions, barring other factors, usually have a normal mentality. Their motor development, of course, is delayed, but on the whole the potentialities for compensation and fair adjustment are far better in this group than in the other two forms of motor defects. Speech, although bearing the characteristic stigmas of dysarthria, is not considerably delayed in its development.

On the whole, these children remain awkward in their movements and are greatly handicapped in competing with normal children, but otherwise find their place fairly successfully in school and society.

For the speech teacher they present a comparatively easy problem, although they retain the characteristics of cerebellar speech, as a rule, for the rest of their life.

This discussion has no claim upon completeness. It may give a faint idea to the teacher of speech of the immensity of the problems presented by the motor defective child. It also may give some grounds for the following conclusions:

CONCLUSIONS.

Any motor defect must be looked upon as the problem of the whole person.

Motor defect in any one of the three central motor systems disturbs tonus and balance also of the other two systems.

Any motor defect interferes with the total function of the central nervous system.

Any defect is irreparable and compensation for it depends on residual functions.

Improvement of functions largely depends on maturation.

Any form of training must have its limitations in the potentialities of the residues.

The teacher of speech must think realistically on this problem and avoid false hopes and unfounded optimism.

Cooperation with the neurologist, physiotherapist, psychologist is essential for best results.

The total personality of the child must be evaluated.

Methods of teaching and training have to be adapted not only to the nature of the motor defect, but also to the individual needs of the child.

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MISSISSIPPI VALLEY MEDICAL SOCIETY "ANNUAL AWARDS."

The Mississippi Valley Medical Society (Headquarters, Quincy, Ill.) has recently established a number of annual awards in an effort to expand the influence of the organization. These are as follows:

1. To encourage recent medical graduates to promptly become interested in post-graduate study, the Society will award each year five free memberships to recent graduates from each of the recognized medical schools of Illinois, Missouri and Iowa. These memberships will be good for two years and will be determined by the deans of the respective schools on the basis of school scholarship, it being required that the appointees be licensed to practice medicine in Illinois, Missouri or Iowa.

2. A silver and a bronze medal will be awarded each year for the most interesting scientific exhibits at the annual meeting.

3. A cash prize of $100.00 and gold medal will be awarded annually for the most interesting contribution to be submitted on a subject determined by the "Annual Awards Committee." This contribution must be of interest to the physician in general practice, and the winner will be invited to present it before the Society at its annual meeting.

4. The Society also has under consideration the annual award of a gold medal to one of its members(?) for distinguished services rendered the profession. This will be determined by the Board of Directors at its next meeting (September).

The matter of annual awards will be in charge of a Secret Committee of five Society members appointed by the President.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY.

Meeting of May 19, 1937.

Radiographic Device for the Anteroposterior Mensuration of the Ethmoids and Sphenoids. Dr. Solomon Fineman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Carcinomatous Metaplasia in Case of Uncontrollable Polyposis of the Nose. Dr. I. B. Goldman.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Some Cases of Special Interest to the Otolaryngologist Seen on the Neurological Service. Dr. Page Northington.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Frontal Lobe Abscess Secondary to Empyema of the Sphenoid. Dr. Abraham Kaplan. (By invitation.)

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

Orbital Infections Due to Nasal Sinusitis. A Study of 114 Cases. Dr. Louis Hubert.

ABSTRACT.

Infections from the nasal sinuses may extend into the orbits in two ways, namely: 1. by the bony orbital walls which separate these sinuses from the orbital cavities; 2. by the venous blood stream; *i.e.*, the veins of the various sinuses which anastomose with the superior and inferior ophthalmic veins in the fatty cellular tissue of the orbits.

CLASSIFICATION OF THE ORBITAL INFECTIONS.

1. Inflammatory edema of the eyelids, with or without edema of the orbit: 31 cases.

2. Subperiosteal abscess: 46 cases. *a.* edema of the lids: 9 cases. *b.* spreading of the pus to the lids: 37 cases (erroneously called orbital abscesses).

3. Orbital abscess: 22 cases.

4. Orbital cellulitis: 11 cases (severe, 9; mild, 2).

5. Cavernous sinus thrombosis: 4 cases (septic, 2; aseptic(?), 2).

INVOLVEMENT OF SINUSES.

1. Frontal sinus	30
2. Frontal sinus and ethmoids	22
3. Ethmoids	26
4. Ethmoids and antrum	5
5. Antrum	2

6. Ethmoids and sphenoid..... 1
7. All sinuses..... 4
8. Sinus involved not certain..... 24

TREATMENT.

1. Edema of eyelids with or without edema of the orbit: nonoperative, conservative treatment.

2. Subperiosteal abscess. *a*. Preliminary: curvilinear incision as used in the external operation, separation of the periosteum, examination of the bony orbit and evacuation of the pus. *b*. Radical operation after subsidence of acute symptoms.

3. Orbital abscess. Exposure of orbital wall and periosteum as in No. 2. If both are intact an incision is made into the orbital fascia and pus is drained. If the bony wall is diseased a radical operation is done after subsidence of the acute symptoms.

4. Orbital cellulitis: In mild cases nonoperative treatment. In severe cases, same as No. 3, but as pus is not found, a radical operation is done immediately to decompress the orbit and so prevent extension to the cavernous sinus and to the meninges, and to relieve pressure on the optic nerve.

5. Cavernous sinus thrombosis: Preventive measures.

DISCUSSION.

DR. FRANK C. KEIL: Dr. Hubert has left very little to discuss in his excellent study of 114 cases of orbital infections due to nasal sinusitis. There are a few points in diagnosis and treatment which deserve special attention: 1. Differential diagnosis. 2. Fundus examination. 3. Some points in operative treatment to be emphasized.

Although various observers have found that over 50 per cent of orbital infections are secondary to sinus disease, it is well to remember other etiological factors and that a coexisting sinus infection might not be the causative factor. Although serological and X-ray examinations along with local signs in the nose will aid in eliminating tuberculous, luetic and neoplastic conditions in the orbit, other conditions must be eliminated. The spread of infections from neighboring parts, such as an acute dacryoadenitis, acute dacryocystitis, hordeolum and a panophthalmitis must be considered. Infected wounds of the face, erysipelas, infections secondary to septic pharyngitis, abscessed teeth and metastatic infections from a septicemia or a septic venous thrombosis in the extremities should be noted. The latter conditions might also be responsible for a tenonitis, which must be differentiated.

Exophthalmos, redness and swelling of the lid in a bottle-fed infant from four to nine months of age should arouse a suspicion of infantile scurvy. Cases have been reported of subperiosteal orbital hemorrhages in scorbutic infants who have been operated for orbital abscesses. A temperature of 102 or 103 frequently found in infantile scurvy is often very misleading.

Edema and redness of the lid, a frequent sign of an acute sinusitis, must not be confused with an exophthalmos. The former does not indicate orbital involvement. The positive signs of orbital involvement are chemosis of the conjunctiva, exophthalmos or proptosis of the globe, along with loss of mobility. Subjective symptoms of pain in the eyeball and orbit, or pain on moving the eyeball, blurring of vision and tenderness of the globe on pressure frequently present, might be absent. It is also possible for an orbital infection of nasal origin to occur with negative nasal findings. The original nasal infection might have cleared before the orbital signs became manifest.

The fundus signs are frequently conspicuous by their absence. The edema of the corneal epithelium usually present renders an ophthalmoscopic examination exceedingly difficult and unsatisfactory.

With thrombosis of the ophthalmic veins or central vein of the retina often found associated with cellulitis of the orbit and cavernous sinus thrombosis, extensive retinal hemorrhages, optic neuritis, papilloedema and venous engorgement are noted early in the disease.

Dr. Hubert has given a very concise pathological classification of orbital infections. It is frequently impossible to assign a case to one of a group, as the conditions frequently overlap. It is only after the incision has been made that the case can be properly classified.

As to treatment: conservative measures might be followed a longer time with adults than with children. The small size of the parts concerned in the latter might lead to immediate extension of the infection to the meninges and other complications.

The old treatment of a puncture with a cataract knife through the conjunctiva into the orbit has been condemned for some time. This procedure gave no drainage and only scattered the infection throughout the noninfected orbital fat. Unfortunately one can find this treatment recommended in some of the not so old textbooks in ophthalmology.

The curvilinear incision is the accepted incision. If pus is not found on separating the periosteum, incisions should be made through the fascia into the cone of ocular muscles. Until this has been done, one cannot be sure that an abscess within the muscle cone has not been overlooked.

In closing, I want to thank the Section of Otolaryngology for the privilege of discussing this interesting paper.

Dr. Francis W. White: The comprehensive paper of Dr. Hubert on orbital infections leaves but little for discussion. Dr. Keil has very carefully elaborated upon the ophthalmological aspects of orbital infections caused by or only incidentally associated with acute suppurative sinusitis. Therefore, it devolves upon me only to stress some of the points concerning classification and treatment that an analysis of the 114 case reports revealed, plus the knowledge gained by the application of these deductions.

However, those of us who have been observing and have noted a lessening of the very radical sinus surgery and the beginning of a more conservative surgery, are no doubt in accord that the latter is the logical method of treatment. Not alone is the improvement in procedure due to a change in the attitude of the rhinologist, but rather to the teamwork of the rhinologist, ophthalmologist and roentgenologist. The latter, particularly, is a most important ally.

You will find the classification of acute sinus diseases as given tonight of the greatest help. The treatment is predicated upon results that cannot be doubted. Probably the type most frequently complicated by surgery is the first; namely, edema of the eyelids and orbit. Too great haste in operating only prolongs a condition that under conservative treatment would recover. The second type, subperiosteal abscess, is self-explanatory and the treatment is logical. It is in the third type, or orbital abscess, that grave consequences may arise if the surgeon does not seek pus by boldly incising the orbital fascia and employ two of the surgical triad of pioneer surgeons, which was: "Open, disinfect and drain." We do not resort to disinfection, however, as heretofore. In the fourth type, namely, orbital cellulitis, although the condition may clear up under conservative treatment, still if no improvement is seen or the reverse occurs, then open and drain by incising the orbital fascia. Do not be disappointed if macroscopic evidence of pus is lacking—it is there, and free drainage is your best friend. By removing the adjacent bony wall a decompression of the orbit is obtained. By successfully treating types 3 and 4, type 5—namely, cavernous sinus thrombosis—may be prevented, which is the best form of treatment.

In concluding, may I suggest that you look upon acute suppurative involvements of the nasal accessory sinuses as being *somewhat* similar to pyogenic conditions in the soft tissues of the body. The indications would then be early free drainage of the accumulating pus, and with the recession of the acute symptoms, do a radical removal of all diseased tissue.